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Transitory Migratory Pulmonary Infiltrations Associated With Eosinophilia (Loeffler's Syndrome)*

With the Report of an Additional Case

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Since Loeffler[1,2] in 1932 and again in 1936 described the syndrome which has come to be known by his name, numerous cases of transitory migratory pulmonary infiltrations have been reported in the foreign literature. The English and American literature on the subject, on the other hand, remains remarkably scanty. It may be significant that several of the few cases reported in it occurred in such far-off places as Hawaii and Palestine and not in North America or the British Isles. Whether this apparent distribution of the disease can be accepted at its face value from the standpoint of incidence, or whether the paucity of the English-American literature means unfamiliarity with the syndrome, or lack of alertness in diagnosis, I am naturally not prepared to say.

Freund and Samuelson[3] in 1940 collected from the world literature 105 cases, including the 51 reported by Loeffler in his second communication in 1936. My own search of the American and English literature has revealed considerably fewer than 25 cases, even if all reported cases should be accepted as authentic. Certain of them seem of very doubtful validity, and a count derived only from titles listed in the *Quarterly Cumulative Index Medicus* would be most misleading, as a study of individual cases shows. I am inclined, for instance, because of the lack of serial observations, to question the 4 autopsied cases reported by von Meyenburg.[4] Equally doubtful is the case reported by Smith and Alexander,[5] which concerns a child who never completely recovered from bronchopneumonia and who died of terminal sepsis, possibly associated with leukemia, though even autopsy did not establish the diagnosis.

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It is not surprising, as Freund and Samuelson point out, that Loeffler's syndrome has been recognized only recently, for only recently have mass roentgenologic examinations been made in outpatient clinics, schools, and elsewhere. Discussion of the disease is still not definitive, nor is it likely to become so, these same authors observe, until cases begin to be reported in such detail that adequate critical analysis is possible. Detailed data were available in only a quarter of the 105 cases which they collected, and their advice is sound that physicians who encounter possible cases should take systematic histories, including age, sex, season, climate, site of the disease, symptomatology, clinical signs, roentgenologic studies, blood studies, sputum examinations, stool examinations, duration of the illness, and possible etiologic factors.

Clinical Factors—Loeffler's syndrome is best described as a transitory, migratory pulmonary infiltration demonstrable by roentgenologic examination and associated with eosinophilia, which may be very marked. The severity of the roentgenologic findings and the level of the eosinophilia are frequently in startling contrast to the physical findings, which may be entirely normal and which seldom consist of more than a few moist and sibilant rales over the areas of consolidation.

The clinical course is characteristically mild and the symptoms are minimal. As listed by Loeffler, they include only mild acoustic symptoms, fatigue, and coughing, sometimes accompanied by sharp pains in the chest and possibly by a small production of sputum. More recent writers have added to the list asthmatic attacks, bronchitis, and moderate temperature elevations. Douady and Cohen (cited by Baker[6]) reported a case in which hemoptysis was a feature; tuberculosis was not proven and recovery was rapid.

Kartagener[7] described a case which he classified as Loeffler's syndrome and in which chronicity was an outstanding feature. He also mentioned another type of syndrome described by Lohr and by Leon-Kindberg characterized by symptoms so acute and severe as to suggest a septic process; the illness lasted for several months. Kartagener raised the question as to whether these manifestations were variants of the same disease or, as seems more likely, separate entities. In the two cases reported by Karan and Singer[8] the symptoms were pronounced and in one instance cardiac manifestations were noted; the pulmonary infiltrations as observed serially were also slow in disappearing.

Roentgenologic Findings—Loeffler's original description of the roentgenologic findings, supplemented by Breton's later description (cited by Hoff and Hicks[9]), is to the effect that large or small consolidations appear suddenly in various parts of the lung, and disappear rapidly, only to reappear elsewhere within variable pe-

riods of time. In Loeffler's cases the duration was from 3 to 8 days, and in one instance there was a recurrence at the end of a year. The infiltrations are most frequently observed in the lower lung fields, near the diaphragm, and the shape is various. They may be large or small, unilateral or bilateral, irregular or circular, and sharply defined or vaguely outlined. They sometimes bear a distinct resemblance to the adult type of pulmonary tuberculosis.

Eosinophilia—Although eosinophilia, sometimes accompanied by a moderately high leukocytosis and sometimes by a slightly elevated sedimentation rate, is one of Loeffler's original criteria of diagnosis, in many of the reported cases the eosinophilic percentage was low, and sometimes eosinophiles were absent from the blood. Soderling's[10] explanation is that eosinophilia is not marked when infection and fever are present, but tends to reappear when these manifestations have subsided. The variability has also been explained by the variability of the supposedly responsible protein allergens. Whether this disregard of Loeffler's original criteria of diagnosis is justified is open to question; if it is not, the number of reported cases in the English-American literature must be still further reduced. There is no parallelism, Loeffler pointed out, between the size of the roentgenologic lesion and the degree of eosinophilia, which may be at its highest level when the pulmonary infiltrations have begun to diminish.

Pathogenesis—The most confused factor in Loeffler's syndrome is the pathogenesis, which is still undetermined and which is apparently multiple. Loeffler himself concluded that the pulmonary infiltration was on an allergic basis, with the pathogenesis similar to that of erythema nodosum. He considered, and ruled out, pulmonary embolism with infarction, pneumonia, bronchial asthma with atelectasis, pulmonary tuberculosis, and ascariasis. Maier,[11] who studied 100 cases personally, regarded the infiltration in this disease as identical with the temporary infiltrations long recognized in asthma, in which eosinophilic pneumonia is the background of the pathology. This type of pneumonia has been fully described by Miller and his associates,[12] who do not, however, regard the condition as the same entity as the disease described by Loeffler.

Soderling explained the lung picture as due to stagnation of secretions in bronchitis, which 4 of his 5 patients presented, combined with bronchial spasm and resulting in localized areas of atelectasis and emphysema. Such localized areas, he pointed out, may give rise to parenchymal shadows in severe attacks, although the findings are always transient. Breton (cited by Baker) advanced the same opinion. Soderling also believes that the Loeffler syndrome may be the real explanation of many cases of atypical pneumonia and abortive pneumonia, as well as of many cases of supposed

tuberculosis in which recovery takes place rapidly.

Engel (cited by Soderling) reported an epidemic cough which occurs in the general population in China in May and June, when the privet is in flower. On two occasions when he himself developed such a cough, roentgenologic examination showed massive pulmonary consolidations, associated with eosinophilia of 20 to 25 per cent. The pulmonary infiltration disappeared within 24 hours in the first attack and within 6 days in the second. A similar morbid picture was observed in another patient, whose eosinophilia, however, was only 6 per cent. Engel conceived of the lung changes as a Quincke's edema and proposed for the disease the name "oedema allergicum pulmonis." Soderling regards Engel's observations as next in importance to Loeffler's and spoke of the disease as the Loeffler-Engel syndrome. Other writers have accepted them less wholeheartedly.

It is now generally believed that the disease develops on an allergic basis. In most cases, however, the etiologic agent (or agents) responsible have not been identified, though intestinal parasitism, with apparent reason, has most frequently been indicted.

Opinions differ as to precisely how the pathologic changes are brought about. Muller (cited by Frimodt-Moeller and Barton[13]) was able to produce in himself multiple fleeting pulmonary infiltrations, with associated eosinophilia, by eating material containing *Ascaris* ova and suggested that the roentgenologic shadows may be caused directly by the passage of *Ascaris* larvae through the lungs, after they have penetrated into the liver through the intestinal wall. The possibility of such a migration has been demonstrated by Japanese workers. Another theory, which on the surface seems more reasonable, is that the pulmonary changes are indirect and represent an allergic reaction to the presence of ascarids in the body. It is acknowledged that reactions of this sort may be violent. Baer,[14] for instance, mentioned a zoologist of his acquaintance who developed severe rhinitis and conjunctivitis if he so much as walked across a laboratory in which ascarids were being worked on.

Cause and effect reasoning also substantiates the theory that parasitism is responsible for Loeffler's syndrome. Stefano (cited by Hoff and Hicks) reported a case of recurrent asthmatic attacks associated with transient areas of pulmonary infiltration, in which amebae were found in the sputum though not in the stools. Emetine therapy cured the infestation and the patient was simultaneously completely relieved of his asthma. Beck[15] reported a clearcut case of Loeffler's syndrome in which he observed dramatic clinical improvement, disappearance of urticaria, and regression of the pulmonary infiltration following treatment by crystalloids of intestinal infestation with *Strongyloides intestinalis* and *Ascaris lumbricoides*.

Hoff and Hicks reported a case of this syndrome associated with *Endoemba histolytica* infestation. The patient had had severe asthmatic attacks for 3 months, and eventually presented a clinical picture suggestive of early amebic hepatitis. Although treatment with anayodin resulted in only slight improvement, the response to emetine was dramatic, the patient being completely and simultaneously relieved of his asthma and his intestinal infestation. These authors speculate that if stool and sputum examinations had been carried out in the cases reported by Engel, it is quite possible, in view of the frequency of amebic infestation in China, that amebiasis rather than the privet flower might have been revealed as the etiologic agent.

Diagnosis—The diagnosis of Loeffler's syndrome rests upon three considerations: (1) The radiologic picture, the severity of which is out of all proportion to the insignificance of the physical findings and the mildness of the clinical course. (2) The transience of the roentgenologic findings, the persistence of which raises doubt as to the diagnosis. (3) The degree of eosinophilia, which, as has been noted, cannot be correlated in respect to chronology with the severity of the pulmonary changes as demonstrated by x-ray.

Films must be made at frequent intervals, for diagnosis cannot be made upon a single investigation. Loeffler's advice that examinations be made every second day represents an ideal rather than a practical plan. Baer notes that a single film in his case suggested neoplasia and others have called attention to the similarity between the roentgenologic picture in this disease and in coccidioidomycosis, as described by Dickson.[16] Tuberculosis, however, presents the greatest difficulty in differential diagnosis. It is excluded by the variations in the roentgenologic shadows, the prompt disappearance of the pathologic changes, and the consistent failure to find acid fast bacilli in the sputum. Indeed, the categoric statement may be made that if, after adequate tests, tubercle bacilli cannot be found in the sputum or the gastric secretion, the disease is not tuberculosis.

The differentiation of Loeffler's syndrome from tuberculosis is extremely important. For one thing, the latter disease demands a regimen of life, with all its social and economic implications, which the former does not. For another, the institution of certain forms of treatment for tuberculosis, such as pneumothorax, could readily give rise to disastrous consequences in a non-tuberculous subject.

Therapy—It will be noted that in all the reported cases the therapy was directed toward the allergic manifestations rather than toward the Loeffler syndrome itself, the course of which was practically always so mild that treatment was not required. If therapy should prove necessary, calcium lactate in 3 gm. doses is recommended by Engel (cited by Soderling).

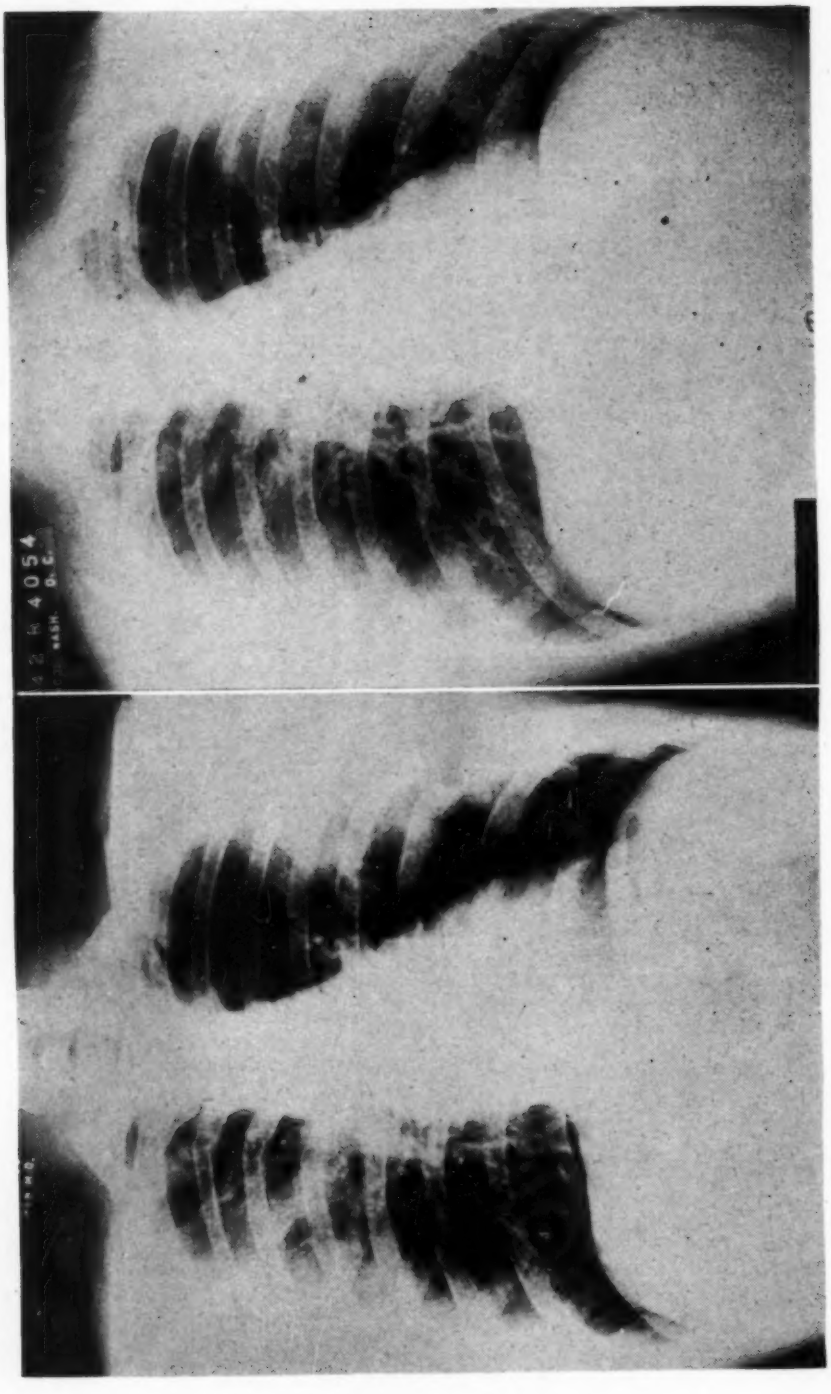

Fig. 2—3/16/42

Fig. 1—3/12/42

CASE REPORT

All things considered, the case reported herewith seems to fit into the syndrome described by Loeffler. The essential facts follow:

M. B., a white male government clerk 21 years of age, was first seen in consultation March 16, 1942. For the past 6 months he had suffered from, and entirely neglected until 2 weeks earlier, "a very bad cold" and moderately productive cough, associated with a slight loss of weight and a sense of fullness and later of soreness in the chest. When he first sought medical advice from his family physician, roentgenologic examination of the chest had revealed an infiltration in both lungs, more marked on the left side, extending from the level of the third rib anteriorly to the level of the fifth rib. The findings had been interpreted as an acute infection in both upper lobes, possibly of tuberculous origin.

The past history was without incident except for the usual diseases of childhood (measles, chickenpox and whooping cough) and appendectomy for acute disease at the age of 9 years. All other illnesses were specifically denied, and the family history was specifically negative for cancer, diabetes, tuberculosis, renal and cardiac disease, and allergic states.

Physical examination at this time was essentially negative. Stereoscopic examination of the chest revealed the bilateral lesions previously described, which were now somewhat less extensive.

Time and space will be saved if certain laboratory examinations which were carried out at this time and during the subsequent 23-month period of observation, and which were always substantially the same, are summarized at this point. Urinalysis was always essentially negative. The sputum, although examined by smear, concentration and culture at various times, never revealed acid fast bacilli, and the only findings of significance was an eosinophilia, which varied from moderate to marked. The tuberculin patch test and the Mantoux test were negative, as were all the skin tests carried out at various times during the illness. Examination of a stool specimen March 16, 1942, revealed larvae of *Ascaris lumbricoides*, but all subsequent stool examinations were negative.

The hemoglobin was never lower than 78 per cent and the red blood cells never numbered less than 4,200,000 per cu. mm. The sedimentation rate varied between 13 and 24 mm. per hour. The white blood cell count and the differential count, however, varied considerably at different periods of the illness. March 16, 1942, the white blood cell count was 14,450 per cu. mm. The differential count showed 48 per cent eosinophiles, 32 per cent segmenters, 17 per cent lymphocytes, and 3 per cent myelocytes.

Fluoroscopic examination April 24, 1942, revealed clear lung fields, but in view of his recent history and the previous roentgenologic findings, the patient was told that he apparently had an active pulmonary infection, probably of tuberculous origin, and continued observation was advised until a positive diagnosis could be made. A contemplated marriage was also advised against.

The patient disregarded both pieces of advice. He was not seen again until July 20, 1942, at which time he was suffering from an asthmatic attack (his first) of such severity that he was hospitalized at once. Dr. James Nolan was asked to investigate the allergic state and has continued to direct the treatment ever since. Stereoscopic examination of the chest at this time again revealed no abnormalities.

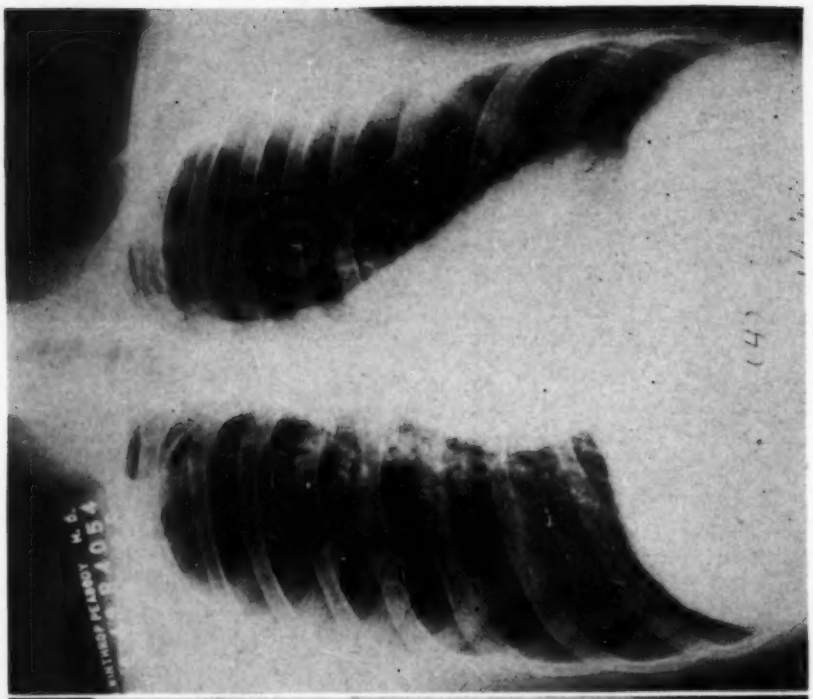

Fig. 4—1/16/43

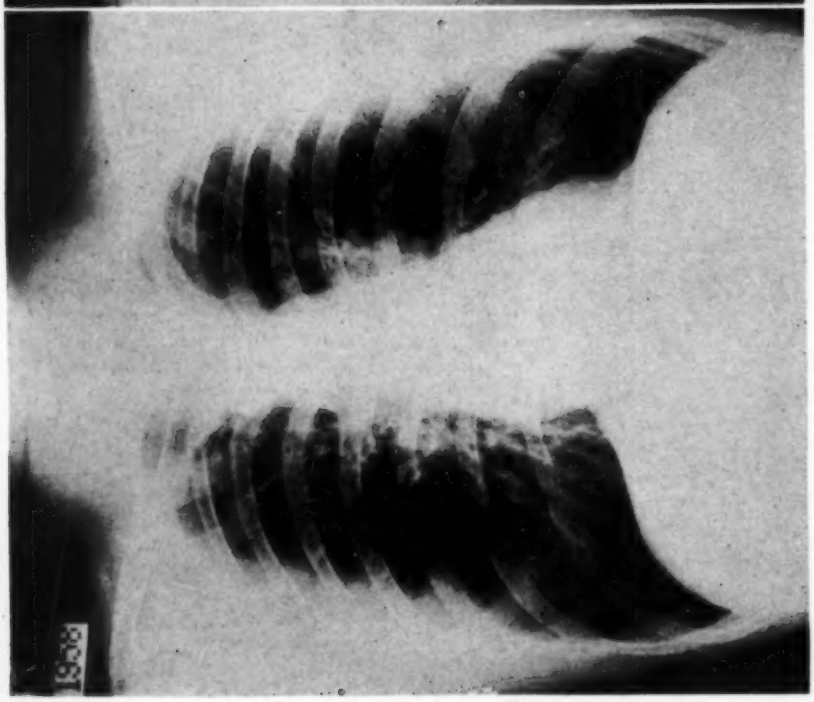

Fig. 3—7/20/42

The patient was discharged from the hospital at the end of a week, but continued to have attacks of asthma, chiefly in the form of wheezing and dyspnea, at first every day or two, then every 6 or 8 hours, and eventually every 2 hours. The only other symptom was anorexia.

Stereoscopic examination of the chest January 6, 1943, again showed the lung fields clear. The white blood cells numbered 14,450 per cu. mm., with 36 per cent eosinophiles, 48 per cent polymorphonuclear leukocytes, and 16 per cent lymphocytes.

The patient was hospitalized for the second time March 7, 1943, for an acute upper respiratory infection and continued attacks of asthma. The temperature ranged between 100° and 102° F. for several days, but there was no other marked elevation during the illness and seldom any elevation at all. Physical examination was negative except for many moist, musical rales throughout the chest, somewhat more marked at the left apex. Psychiatric examination, undertaken because of a mental and emotional strain to which the patient had recently been subjected, furnished no diagnostic aid.

Shortly after his discharge from the hospital the patient went to Florida, where he spent 4 months and where he had no medical supervision. Soon after leaving the hospital he developed anesthesia of the dorsum and palm of the left hand, with paralysis and anesthesia of the fourth and fifth fingers of the same hand, possibly as the result of the adrenalin therapy used to control the asthmatic attacks. The paralysis gradually disappeared, but the patient continued to complain of numbness over the affected areas and of pain along the course of the ulnar nerve. Despite the change of climate his asthmatic attacks continued.

The patient was again seen in consultation August 9, 1943. Stereoscopic examination of the chest August 6 had revealed a homogeneous density overlying the lateral portion of the left lung and extending from the apex to the seventh rib posteriorly. A diffuse, predominantly fibrotic infiltration of the upper lobe on this side enclosed several radiolucent shadows simulating cavities. The heart was somewhat retracted to the left as the result of the fibrotic changes. Another triangular area of dense infiltration was observed in the right upper lobe, between the second and third ribs anteriorly. Both bases were relatively clear.

Repetition of the stereoscopic examination August 9, 1943, revealed essentially the same findings as on August 6. They were interpreted as indicative of thickening of the pleura and bilateral fibroid tuberculosis. A decision as to activity was withheld pending further clinical observation and laboratory investigation.

The white blood cell count August 9 was 17,600 per cu. mm. The differential count showed 14 per cent eosinophiles, 2 per cent basophiles, 2 per cent stabs, 12 per cent lymphocytes, and 70 per cent segmenters. Two days later the white blood cell count had risen to 23,800 per cu. mm. and the eosinophilia to 63 per cent; there were 29 per cent polymorphonuclear leukocytes, 7 per cent lymphocytes, and 1 per cent monocytes. The findings were now interpreted as indicative of a severe infectious process with a marked allergic reaction.

The patient was hospitalized for the third time August 26, 1943, because of the severity and frequency of his asthmatic attacks. In the year since their onset he had lost between 35 and 40 pounds and he now looked very ill, though the physical examination was again essentially negative. Stereoscopic examination of the chest August 27 showed the right lung

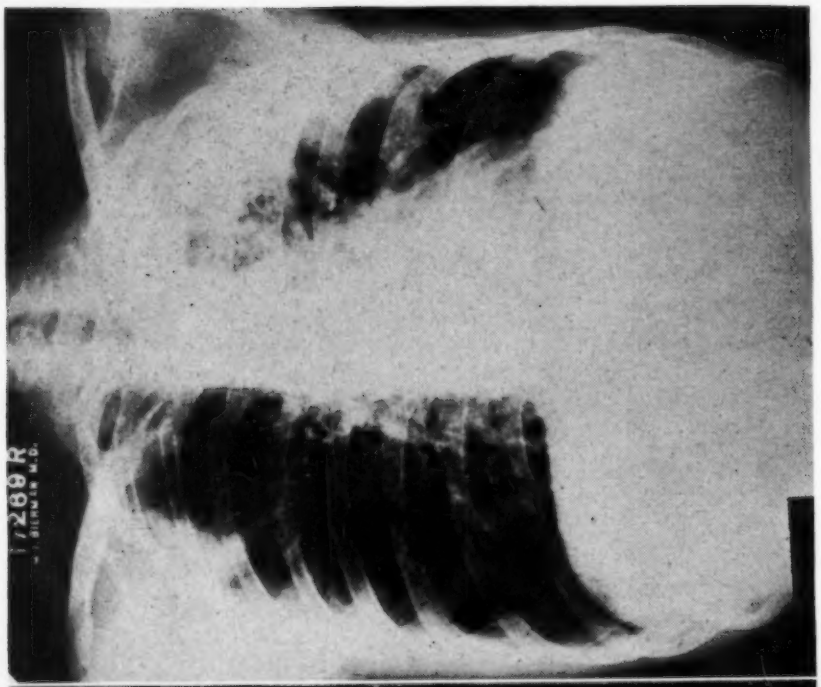

Fig. 6—8/6/43

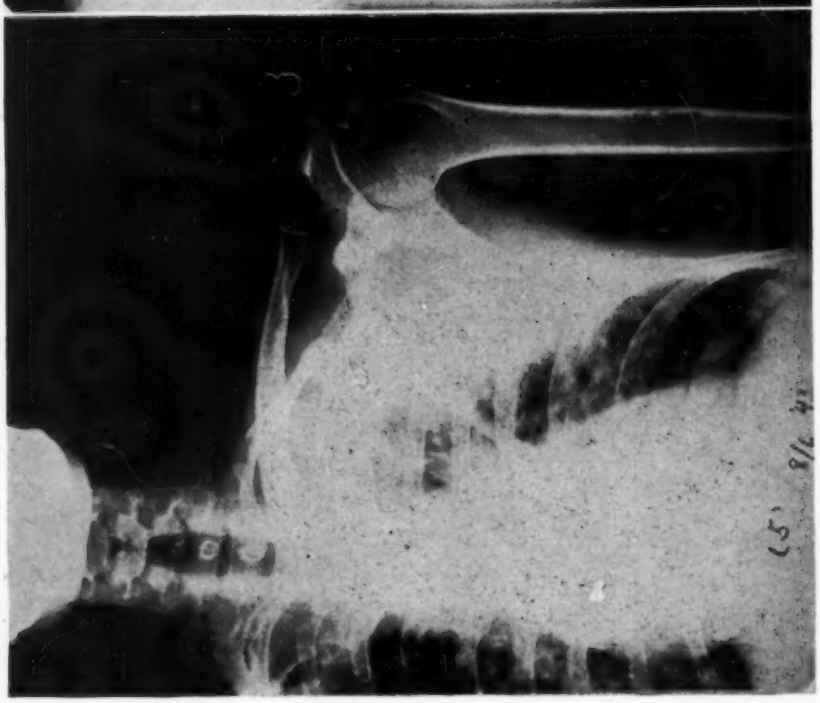

Fig. 5—8/6/43

clear. The entire left lung field was occupied by an extensive fibrotic and infiltrative process, and an area of decreased density in the infraclavicular region was regarded as a possible cavity. Another examination September 3, 1943, showed substantially the same roentgenologic findings, which, although they were suggestive of tuberculosis, in the light of the history and clinical course were now interpreted as probably due to some type of fungous infection.

The differential blood count August 26 showed 61 per cent eosinophiles. September 4 the eosinophilia had fallen to 41 per cent. The white blood cells numbered 15,600 per cu. mm. on the first examination and 11,800 per cu. mm. on the second.

The patient was not seen again until February 14, 1944, at which time stereoscopic examination showed the lung fields clear. The hemogram showed 11,200 white cells per cu. mm., with 19 per cent eosinophiles, 72 per cent segmenters, and 9 per cent lymphocytes.

The patient is still under treatment for asthma, the cause of which, the allergist reports, has not yet been identified in spite of exhaustive tests. At times the chest presents physical findings typical of asthma, but usually, as throughout the illness, the physical examination is essentially negative. Treatment has been entirely symptomatic. Adrenalin gives the most satisfactory results, but they are, of course, always temporary.

COMMENT

There are several striking features in this case:

1) The high degree of eosinophilia, which at one time reached 63 per cent and which was never lower than 14 per cent.

2) The definitely transitory, migratory character of the pulmonary infiltration, which variously involved one lung and both lungs, which varied as to location, and which at intervals disappeared entirely. The changes were sometimes observed within a few days and sometimes at long intervals, though the length of the interims is perhaps not significant, since the patient frequently disappeared from observation. It is worth pointing out in this connection that although the films were made in three different laboratories, the three roentgenologists were in accord in their interpretations.

3) The insignificant character of the physical findings, which, except for certain changes to be expected in severe asthma, amounted to little more than the occasional appearance of rales.

4) The failure on repeated examinations by smear, concentration, and culture, to find acid fast bacilli in the sputum, as well as the negative results of the tuberculin patch and Mantoux tests.

5) The presence of larvae of *Ascaris lumbricoides* in the stool on a single examination but the repeated failure to find them in subsequent examinations over a 23-month period which would seem to exclude this parasite as the etiologic agent in this special case.

My personal role in this case has been that of chest consultant. As such, I have consistently opposed the diagnosis of pulmonary tuberculosis, though I must grant that when I first saw the patient

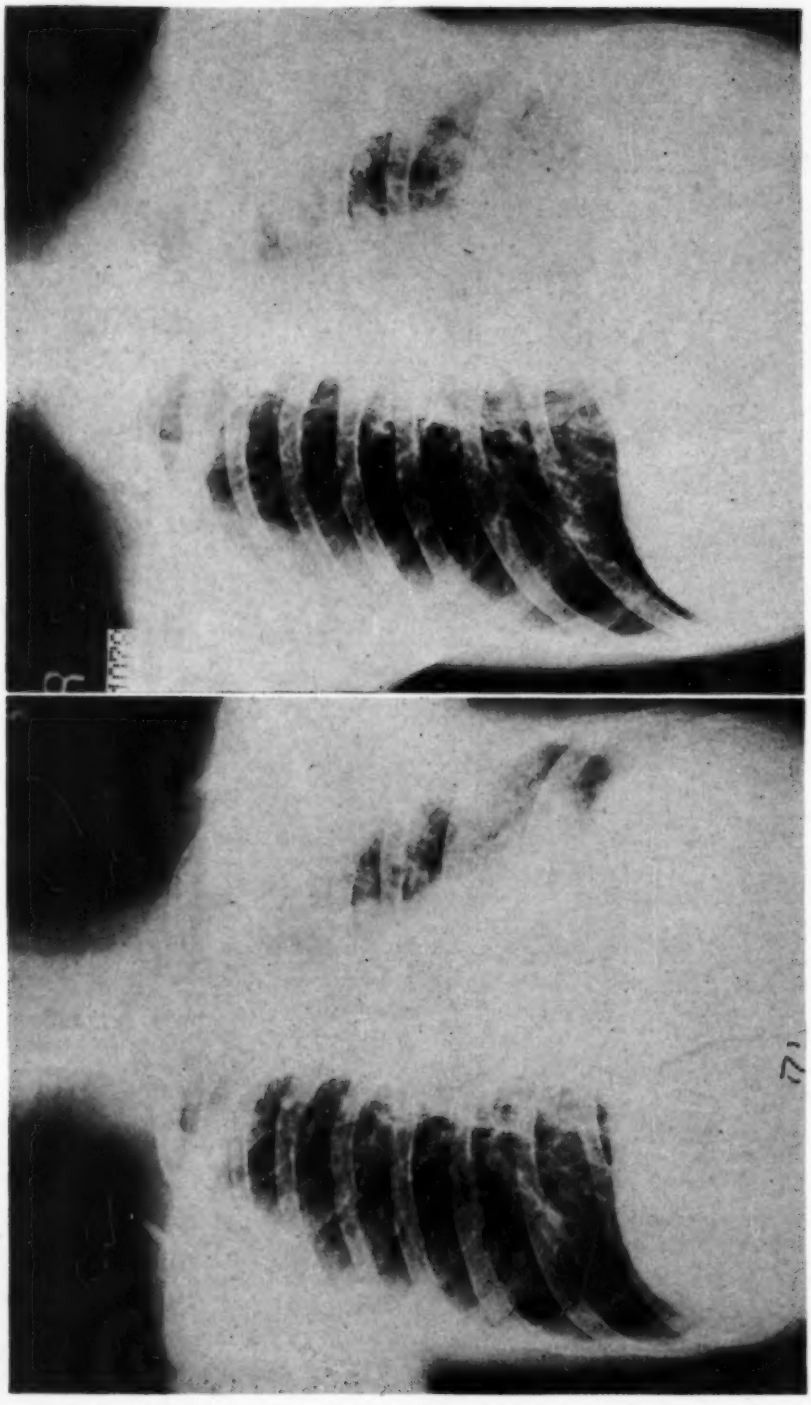

Fig. 8—8/27/43

Fig. 7—8/9/43

the history and roentgenologic findings strongly pointed to this conclusion. My personal conviction, however, is that if acid fast bacilli cannot be demonstrated in the sputum, the disease, whatever it may prove to be, is positively not tuberculosis.

The present impression, as already noted, is that the condition is a marked allergic reaction secondary to some severe infectious process, the cause of which is yet to be determined but the manifestations of which seem to place the case among the few instances of Loeffler's syndrome so far observed in this country.

SUMMARY

1) Since Loeffler's syndrome was first described in 1932, a large number of cases have been reported in the foreign literature and a small number in the English-American literature. Whether all of the reported cases are true instances of the syndrome is open to decided doubt.

2) The syndrome is best described as a transitory, migratory pulmonary infiltration, demonstrable by roentgenologic examination and associated with eosinophilia, which may be very marked. The severity of the roentgenologic findings is in surprising contrast to the physical examination, which is frequently entirely negative, and to the minor symptomatology and mild clinical course.

3) The pathogenesis is still undetermined, but the disease is now generally believed to develop on an allergic background, and intestinal parasitism has been identified as the causative agent in the few cases in which any identification at all has been possible.

4) Diagnosis rests upon the radiologic picture, the transience of the roentgenologic findings, and the degree of eosinophilia. It cannot be made upon a single film. Pulmonary tuberculosis is the most frequent differential diagnostic consideration, and for therapeutic reasons the differentiation is extremely important. Therapy is directed toward the allergic disease, Loeffler's syndrome, in itself seldom requiring treatment.

5) An additional case of Loeffler's syndrome is herewith added to the American literature of the subject.

RESUMEN

1) Desde cuando se describió por primera vez el síndrome de Loffler en 1932, ha aparecido un gran número de casos en la literatura extranjera y un pequeño número en la literatura anglo-americana. Se duda seriamente que todos los casos presentados sean verdaderos ejemplos de este síndrome.

2) La mejor descripción del síndrome es que éste es una infiltración pulmonar transitoria y migratoria, demostrable mediante examen roentgenológico y asociada con eosinofilia que puede ser muy marcada. La gravedad de los hallazgos roentgenológicos presenta

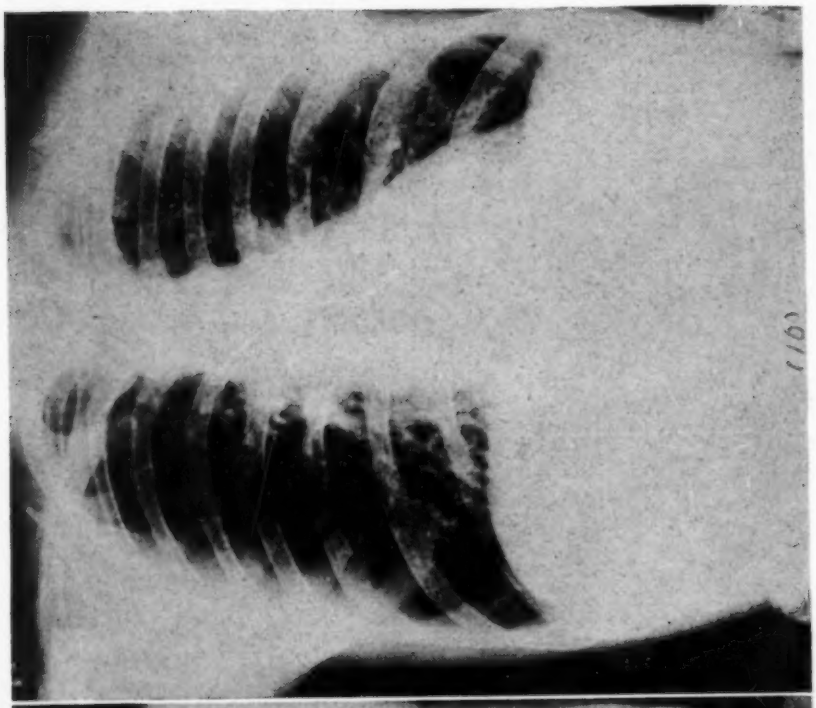

Fig. 10—9/25/43

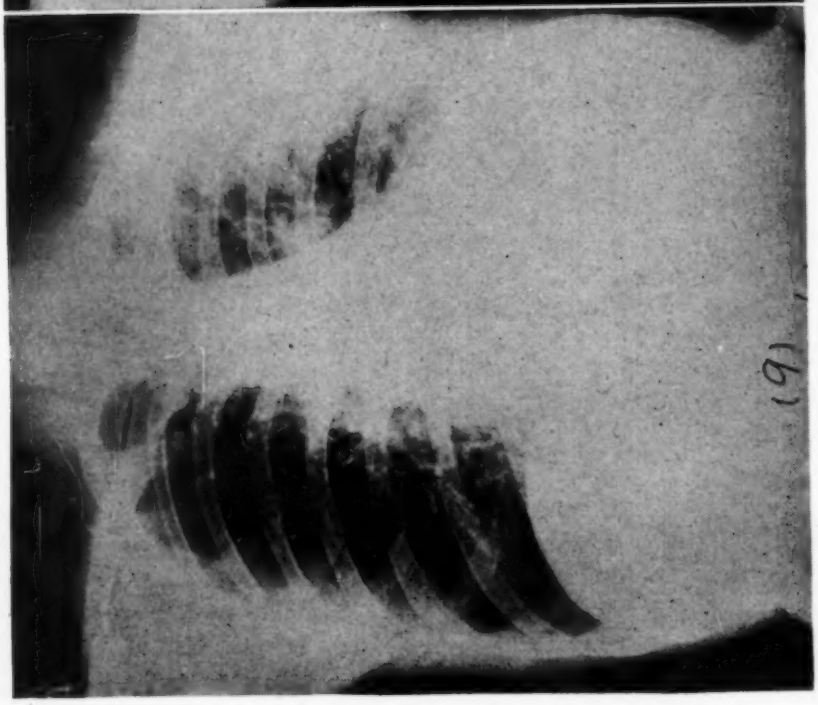

Fig. 9—9/3/43

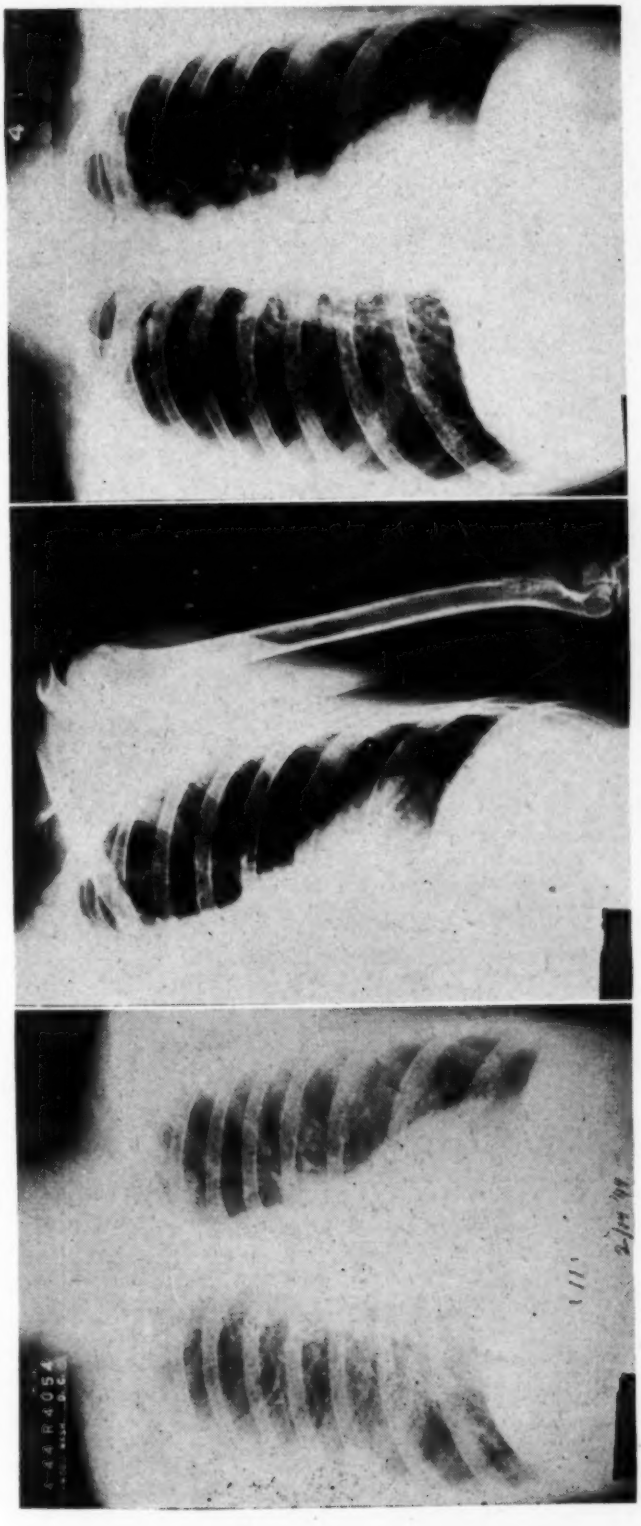

Fig. 13—4/11/44

Fig. 12—4/11/44

Fig. 11—2/14/44

un contraste sorprendente con el examen físico, que con frecuencia es enteramente negativo, y con la sintomatología menor y la benigna evolución clínica.

3) No se ha determinado todavía la patogenia, pero se cree comunmente que se desarrolla en un fondo alérgico y el agente etiológico identificado ha sido parasitismo intestinal en los pocos casos en los que ha sido posible hacer la identificación.

4) El diagnóstico está basado en el cuadro radiológico, en lo transitorio de los hallazgos roentgenológicos y en el grado de eosinofilia. No puede establecerse con una sola película. Se considera más frecuentemente a la tuberculosis pulmonar en el diagnóstico diferencial, y la diferenciación es en extremo importante por razones terapéuticas. Se dirige el tratamiento hacia la enfermedad alérgica; el síndrome de Loffler de por sí, sólo en raras ocasiones requiere tratamiento.

5) Se agrega otro caso del síndrome de Loffler a la literatura americana sobre esta materia.

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Cavernous Breathing: Is There Such a Sound?

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Austin Flint[1] claims the credit of first describing the distinctive characteristics of "cavernous breathing." Flint[2] described cavernous breathing as follows: "The characters of the cavernous respiration are, an inspiratory sound low in pitch, non-vesicular in quality—a simple blowing sound—the expiratory sound still lower in pitch, with the same quality, its length and intensity variable." Flint further stated: "The liability to error is in confounding the cavernous with the vesicular respiration, the chief point of difference being the presence of the vesicular quality in the latter and its absence in the former." Since 1852 the above description of "cavernous breathing" has been accepted by nearly all authors and teachers of physical diagnosis. The following discussion concerns "cavernous breathing" and not the auscultatory diagnosis of cavities in the lung. Is "cavernous breathing" described by Austin Flint truly characteristic of the noise produced in the cavity? Flint qualifies the cavity over which cavernous breath sounds are heard: an empty cavity, freely communicating with the bronchial tree and with flaccid walls. The author has the opinion that the cavity produces no such auscultatory findings. The opinion is based on the fact that the author has never found the distinctive characteristics of cavernous breathing over cavities. Flint has mistaken exaggerated vesicular breathing for evidence of cavitary formation. The mistake is a natural one, for exaggerated vesicular breathing is identical to Flint's cavernous breathing except for differences in the qualities of the sounds during inspiration (Table I).

Flint[2] stated in his textbook published in 1875: "The liability to error is confounding the cavernous with the vesicular respiration, the chief point of differences being the presence of the vesicular quality in normal vesicular respiration and its absence in cavernous respiration." If there is a liability to error between normal and cavernous respiration the opportunity for error is multiplied tenfold in differentiating exaggerated vesicular respiration from cavernous respiration.

How many physicians can answer, right off, in what types of respiratory sounds the quality of the inspiratory sound differs from the expiratory sound? The author has put this question to many classes of medical students year after year in their senior year and with only a few correct answers. Vesicular breathing is the only sound in which the quality of the inspiratory sound differs from the

Fig. 1 *Fig. 2*

Fig. 1—A reproduction of an x-ray film (tomograph) revealing a large superficial cavity in the upper part of the right lung, 4 cm. posteriorly from the thoracic wall. Auscultation over the right upper lobe posteriorly revealed the following: Breath sounds were markedly diminished in intensity and broncho-vesicular in character. Molst rales were distant and high pitched. Here we have a large superficial cavity. The cavernous murmur described by Flint was not heard. *Fig. 2*—A reproduction of an x-ray film (tomograph) taken at 4 cm. from the posterior thorax. The cavity is in the left upper lobe. On auscultation no "cavernous respiration" as described by Flint was heard. Broncho-vesicular breathing with fine high pitched moist rales were heard over the area of the cavity.

TABLE I

		Normal Vesicular Respiration	*Cavernous Respiration*
Inspiration	Quality	Vesicular	Blowing
	Pitch	Low	Low
	Duration	|————————|	|————————|
Expiration	Quality	Blowing	Blowing
	Pitch	Lower	Lower
	Duration	|——— |	|═══ |

expiratory sound. Unfortunately for most medical students and physicians it is difficult to distinguish between "vesicular" and "blowing" qualities. The vesicular quality is difficult to describe. Various terms were in use as early as 1856[3] in describing the vesicular quality—soft, breezy, expansive; a sound produced by a gentle breeze among the branches and leaves of the trees; similar to that of a pair of bellows, the valve of which acts noiselessly; like the sound of softly sipping air with the lips, etc. As usually happens, when description is difficult, we have many. The one least descriptive but most romantic has been universally used to describe the inspiratory vesicular murmur, i.e., the sound produced by a gentle breeze through the branches and leaves of the trees. The author has attempted to find something in common between the sound produced by the wind gently breezing between the branches and leaves of trees and the quality of vesicular breathing, over the past twenty summers, but has failed to associate the two sounds in any manner. He is content just to call the quality of vesicular inspiration a "vesicular quality" and leave to the imagination of the student the description of the sound. All unrhythmic sounds are difficult to describe and differentiate where there are no sharp variations. This unfortunately is true of most breath sounds except the musical one, "amphoric breathing." Once you hear a musical sound you have no further difficulty in remembering the quality irrespective of pitch and duration, as, for example, the sound of piano, violin, harp, etc. Could you *describe* the sounds of these musical instruments? Yet there is no difficulty in *identifying* them. From the above discussion, when two sounds have the same characteristics as have both vesicular and cavernous breath sounds, except-

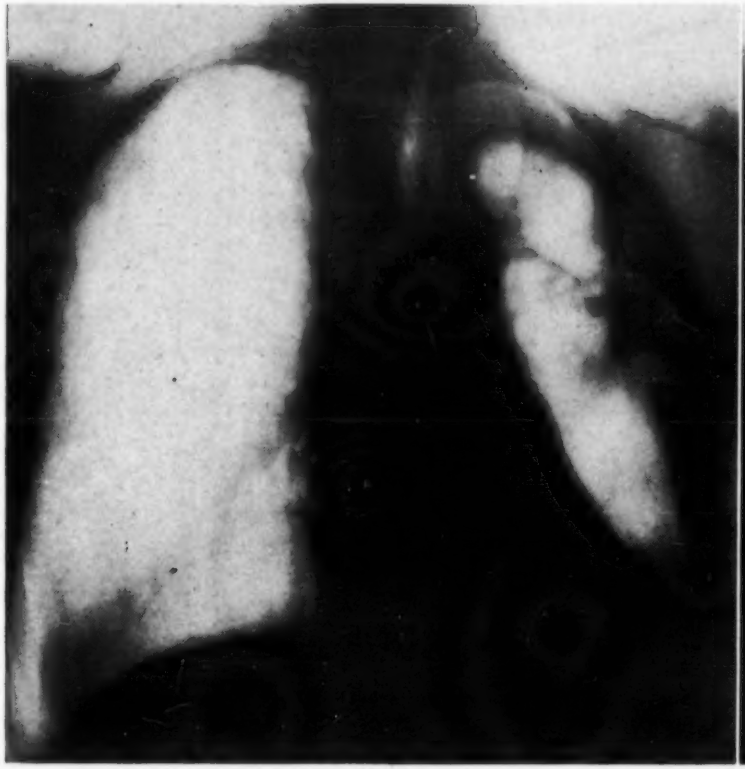

Fig. 3

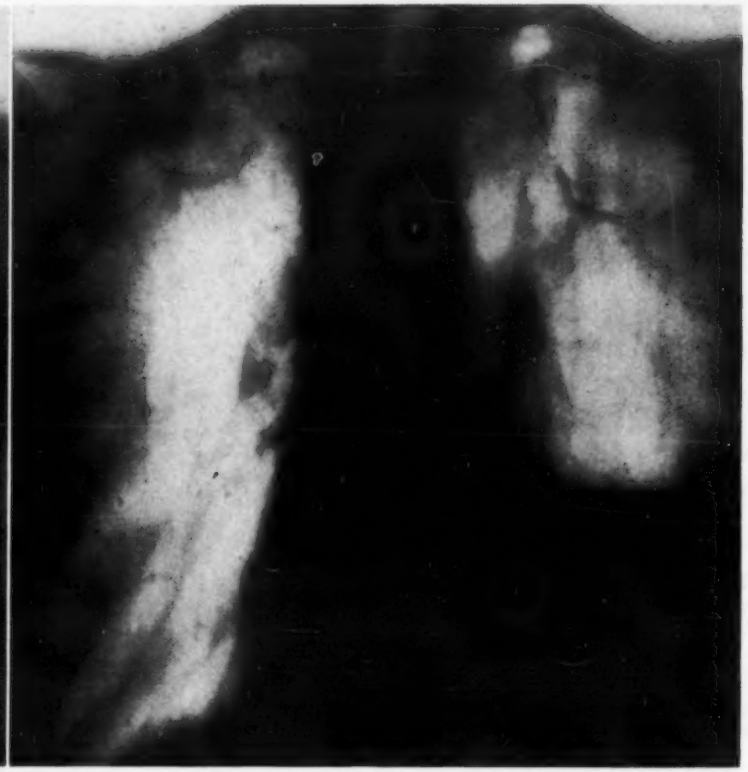

Fig. 4

Fig. 3—A reproduction of an x-ray film (tomograph) showing a giant large cavity in the left lung. The tomographic study revealed the whole cavity to be 12 cm. from the posterior thoracic wall. On auscultation no "cavernous respiration" as described by Flint was heard. The breath sounds over the upper half of the left lung were diminished in intensity and broncho-vesicular in character. No moist rales were heard. *Fig. 4*—A reproduction of an x-ray film (tomograph) taken 6 cm. from the posterior thorax. In the left lung there is a giant superficial cavity and smaller cavity in the right upper lobe. Over both cavities the breath sounds were diminished in intensity and broncho-vesicular in character. High pitched moist rales were also heard. "Cavernous respiration" as described by Flint was not heard over both cavities.

ing slight variations in the quality of inspiration, we can understand the possibility for error.

When we further consider that Flint described cavernous breathing in 1852, when one worked without the aid of fluoroscopic and x-ray examinations, we can understand that errors in diagnosis could easily be made. How could Flint prove that cavitary formation existed in the lung over which he elicited cavernous breathing? The proof must have been in the morgue. When death is due to pulmonary tuberculosis, there is little trouble in finding a cavity in the lung and it was the latter type of confirmation that was used to prove cavernous respiration.

It is the author's concept that Flint made the error of interpreting as cavernous breathing the form of exaggerated vesicular breathing frequently found in the vicinity of tuberculous infiltration.

Flint[3] himself states: "An exaggerated vesicular murmur does not proceed from the portion of the lung affected but from the healthy lung situated near or remote from the seat of the disease." Fournet[3] also had similar thoughts of vicarious vesicular murmur. Fournet pointed out that exaggerated respiration ensues in healthy lung situated in the immediate vicinity of a local affection which compromises or abolishes the function within a limited space. Fournet gave as an example: surrounding a mass of tubercle, the vesicular murmur is rendered unduly intense. Not a bad observation for Flint and Fournet in 1850.

The author remembers, between 1920 and 1925 when few hospitals and tuberculosis sanatoria made use of the fluoroscope and x-ray, how frequently cavities were charted over the upper lobes anteriorly, the compensatory exaggerated murmur present being mistaken for cavernous breathing. With the increase in use of the fluoroscope and the x-ray the anterior cavities disappeared. We know how infrequently tuberculosis occurs in the anterior portions of the lung. Most cavities are in the posterior segment of the upper lobes and in the apices of the posterior segments of the lower lobes with the anterior segments enlarging and compensating for the loss of function in the posterior segments of the lobes.

Furthermore, the first auscultatory signs of cavity formation would be a combination of broncho-cavernous breathing if there were such a murmur as "cavernous breathing." Before the cavity develops there must be an area of caseation followed by liquefaction. When the liquefied material empties into the bronchial tree, a space forms in the lung that assumes a spherical shape because of the difference in pressure between the bronchial tree and the pleural cavity. The author has never heard cavernous breathing as described by Flint over such cavitary formation. When breath sounds were heard over such cavities a broncho-vesicular or bronchial

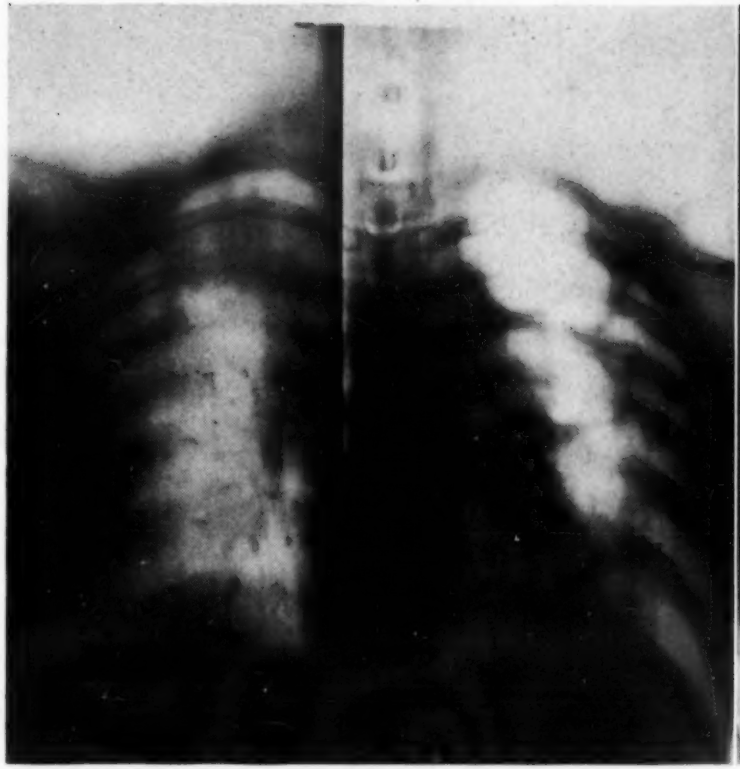

Fig. 5

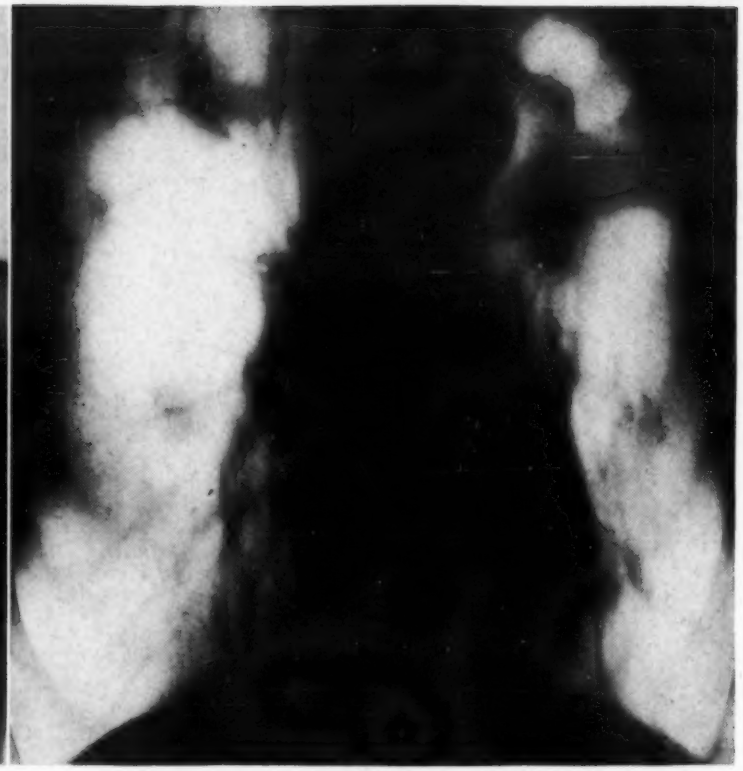

Fig. 6

Fig. 5—A reproduction of an x-ray (tomograph) taken at 4 cm. from the posterior thorax. The tomographic study reveals two superficial giant cavities in the left lung. The lower cavity has a fluid level. No "cavernous breathing" as described by Flint was heard. Posteriorly from apex to eighth rib the breath sounds were markedly diminished in intensity. The character of the sounds could not be determined. In the upper half of the left axilla diminished broncho-vesicular breathing and medium high pitched moist rales were heard. *Fig. 6*—A reproduction of an x-ray (tomograph) taken at 10 cm. from the posterior thorax reveals cavitary formation in both lungs. Note the large cavity in an atelectatic left upper lobe. The left major bronchus can be distinctly seen. The cavity is distended in an atelectatic lobe because of a check-valve mechanism. No "cavernous breathing" as described by Flint was heard over the cavities. Over the right cavity broncho-vesicular breathing is heard. Over the left upper lobe the bronchial sound in the major bronchus is transmitted through the atelectatic left upper lobe.

sound was heard. The author agrees with the contemporaries of Flint[3] (Walsh, Skoda, Bath and Roger), with whom Flint debated the finding of cavernous breathing against the finding of a bronchial element over cavities. When the cavity takes on the form described by Flint (flaccid walls, empty and communicating with the bronchial tree), usually diminished breath sounds are heard. Not once has the author heard cavernous breathing, no matter how near such a cavity was to the lung surface. Over that portion of the lung, or lobe or segment of a lobe, which the cavity occupied, the breath sounds were greatly diminished in intensity. In few instances when the cavity lay away from the surface of the lung, the lung tissue overlying the cavity compensated, and exaggerated vesicular murmur was heard. Most cavities have a check-valve mechanism and are distended. It is natural that only small amounts of air enter the cavity and less air escapes. The small amount of air entering and returning from the cavity produces such weak sounds that they are not audible. In the check-valve variety of cavity[4] the overdistention of the cavity compresses the surrounding uninvolved lung tissue and further cuts down on the intensity of the sound. Silent cavities are no new thought, for other workers have previously reported such findings.[5] When the lobe or segment is completely involved with a caseous tuberculosis, there is an immediate loss in the volume of the lung involved. Only part of the caseous area may undergo liquefaction and the cavity may be surrounded with caseous tuberculous disease. The first auscultatory sign is diminished bronchial breathing. The trachea and major bronchi may shift toward the lobe or segment in which the loss of volume occurs. As time elapses, the fluids in the caseous area are absorbed and replaced by fibrotic tissues and calcium. A check-valve cavity may persist in such a caseous area for a long period. The atelectatic area may then become a good transmitter of sound and transmit bronchial or tracheal noises. This sound is frequently and incorrectly called broncho-cavernous breathing. Certainly the low pitched sound resembling "vesicular breathing" that Flint named "cavernous breathing" is not heard in conditions described above.

The following figures of x-rays of cavities of the lung will illustrate the absence of Flint's cavernous breathing in the presence of cavities.

From the above discussion the author believes he has built up a case against the teaching of "cavernous breathing" as a sound produced by cavities. The author hopes this presentation will stimulate further investigation of the breath sounds heard over cavities. He further believes that in the teaching of physical diagnosis, the diagnosis of cavity should be emphasized. The student

should be acquainted with the facts that diminished broncho-vesicular breathing is the most common finding over the cavity and that diminished broncho-vesicular breathing accompanied by high pitched moist rales is frequently heard over cavities. Students should also be instructed that thin-walled check-valve cavities are frequently silent. Uninvolved lung tissue overlying cavities may produce vesicular breath sounds. The student should also be instructed that a cavity may be present in an old atelectatic lobe, which may transmit the tracheal or bronchial noises. Such sounds have been frequently called broncho-cavernous breathing, incorrectly. The author, not to confuse the issue of cavernous breathing, has discussed only the auscultatory findings over cavities.

SUMMARY

The author has made the observation over a period of twenty years that the sound described by Flint and named by him "cavernous breathing" is not heard over cavities.

The author believes the sound Flint described was vicarious vesicular breathing, which he incorrectly associated with noises produced by the cavity.

RESUMEN

El autor ha observado durante un período de veinte años que el ruido descrito por Flint y llamado por él "respiración de tipo cavernoso," no se oye encima de las cavernas.

El autor cree que el ruido que Flint describió fue respiración vicaria, que él asoció erróneamente con ruidos producidos en la caverna.

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The Treatment of Bronchial Lesions by the Inhalation of Nebulized Solution of Sodium Sulfathiazole*

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A group of respiratory disorders, apparently refractory to other forms of therapy, was treated by the direct application of sulfa drugs to the mucosa of the tracheo-bronchial tree. This was accomplished by means of the inhalation of nebulized solutions.

The procedure of nebulization has been employed previously, notably with broncho-dilating drugs in bronchial asthma. Recently sulfa drugs have been utilized experimentally and clinically in a similar manner. Biancani[1] has experimentally demonstrated that the inhalation of nebulized solutions results in the deposit of the drug throughout the tracheo-bronchial tree and within the alveoli. Harris et al[2] administered sulfonamide micro-crystals by inhalation and were able to reduce the mortality in mice due to pneumococcus infection. Barach et al[3] reported the experimental use of nebulized promin in the treatment of tuberculosis in guinea pigs and revealed a satisfactory trend and higher survival rate of the treated animals. Castex et al[4] have described the use of nebulized solutions of sulfonamides in respiratory disorders and have noted favorable results.

MATERIAL AND METHOD

Prior to the institution of nebulization, the various forms of therapy employed in 50 consecutive cases without adequate response included sedation, expectorants, iodides, broncho-dilating drugs, bronchoscopy, lipiodol instillation, oral sulfonamides, clearance of upper respiratory foci and vaccines. Each case was selected upon the basis of the following criteria: (1) Symptoms of cough and expectoration of more than six weeks' duration. (2) No response to previous modes of therapy. (3) The presence of a bronchial lesion of bacterial origin. In several instances the non-infectious type of asthma was treated for the purpose of a control sample.

The patients were chiefly ambulatory or semi-ambulatory, white, male, adults, employees of the Panama Canal or in the military service of the United States. In addition to the usual hospital routine work-up, special studies were made in a large percentage by the allergy and nose and throat departments. All cases had sputum

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examinations and roentgenograms of the chest. In many patients blood specimens were submitted after four to seven days of treatment for the determination of sulfathiazole levels.

The method was similar to the one used by Stacey,[5] who reported beneficial results in five cases of bronchiectasis. A 5 per cent solution of sodium sulfathiazole was placed in a nebulizer and this was connected by a rubber tube to an oxygen tank equipped with a flow meter. A rate of flow of four liters per minute was found satisfactory. The patient held the nozzle of the nebulizer between the teeth and breathed with his mouth open for a period of twenty minutes. Treatments were administered three times daily for an average of ten consecutive days. It was determined that approximately 2 cc. of the solution was utilized in a single treatment. Inasmuch as the possibilities of decomposition, irritability and diminution of therapeutic efficiency arose after 48 hours, fresh solutions were dispensed frequently.

RESULTS

The diagnosis in each case was founded upon the correlation of the clinical, roentgenological and laboratory data. The response to therapy was recorded on the basis of reduction in paroxysms of cough, diminution and change in character of expectoration, subsidence of objective signs (wheezes, rales) and general improvement. Table I furnishes the details of the results.

There was no improvement in 7 cases (14%), slight improvement in 11 cases (22%); moderate improvement in 15 cases (30%), and marked improvement in 17 cases (34%). Thus improvement was noted in 43 cases or 86 per cent of the entire series. Generally after three or four days of treatment a definite change was noted; paroxysms of cough lessened, expectoration decreased and the sputum became less purulent. The patient often claimed that breathing seemed easier and the feeling of tightness of the chest disappeared. In the asthmatic group musical rales became scanty or absent and vital capacity tests, as studied in several cases, revealed a definite increase in respiratory reserve.

In two cases toxic reactions were observed and the treatment was abandoned. Both occurred within twenty-four hours and were local, characterized by discomfort and swelling of the naso-pharyngeal mucosa. They were mild and subsided within twelve hours. The fact that no systemic reactions were detected can be explained by the small dosage and subsequently the minimal absorption of sulfathiazole in the blood.

The sulfonamide comparator described by Churg and Lehr[6] was employed in fifteen cases, but was discarded because of inaccuracy for low levels. The method of Bratton and Marshall[7] was utilized in

TABLE I

RESPONSE TO THERAPY*

	Number of Cases	*No Improvement*	*Slight Improvement*	*Moderate Improvement*	*Marked Improvement*
Asthma, Bronchial, Infectious	12	1	3	4	4
Asthma, Bronchial, Non-Bacterial	4	3	1	0	0
Bronchitis, Subacute, Post-pneumonic	8	0	1	3	4
Bronchitis, Secondary to Sinusitis	8	1	1	3	3
Bronchitis, Secondary to Upper Respiratory Infections	9	0	1	2	6
Bronchitis, Chronic with Pulmonary Emphysema	4	1	1	2	0
Bronchiectasis	5	1	3	1	0
Total	50	7	11	15	17

*No Improvement.

Slight Improvement: Satisfactory trend but not symptom free.

Moderate Improvement: Absence of findings except for occasional irritative cough and rales.

Marked Improvement: Total absence of all subjective and objective findings.

fifteen other cases. The majority of readings were negative or showed only traces and the highest level was recorded as 0.3 mgs. per 100 cc. of blood. Blood for study was usually drawn between the first and second nebulizations on the fifth day of treatment. As excretion occurred at a rate which easily counterbalanced absorption, no cumulative effect was observed.

CASE REPORTS

Several illustrative case reports representative of this diverse group are briefly presented:

Case 1—A white enlisted man in the U. S. Army was admitted to the hospital with a history of a chronic cough for the past several years. Paroxysms were moderate and the sputum was muco-purulent in character but not malodorous. A clinical survey revealed a cylindroid type of bronchiectasis of the left lower lobe. Lipiodol instillation, bronchoscopic

aspiration, vaccine, and other forms of therapy were ineffectual. After two weeks of nebulization the paroxysms of cough were less frequent and less severe, and the sputum was more mucoid and less copious. The effect was by no means curative but there was moderate improvement.

Case 2—A white male adult, soldier, was admitted with a history of recurrent attacks of cough, dyspnea, and wheezes. Sputum cultures and roentgen studies were negative. An allergy work-up indicated multiple sensitivity to pollens of grasses, trees and dust. He was considered a case of non-infectious bronchial asthma and did not respond to sulfa nebulization. This is the type of bronchial asthma which is unaffected by the treatment, in contradistinction to the infectious type. He was then placed on a course of specific desensitization and improvement was marked.

Case 3—A white male adult, civilian employee of the Panama Canal, presented a history of several asthmatic attacks following upper respiratory infections during the past several years. Each episode lasted for a period varying from six to ten weeks. All previous forms of treatment, including ephedrine sprays, hypodermic injections of adrenalin, sedation, iodides, and cold vaccine failed to abort or influence the course of the attacks. He was admitted to the hospital on the third day of this last bout and after seven days of nebulization there was a complete disappearance of all subjective and objective findings. Streptococcus hemolyticus, micrococcus catarrhalis, staphylococcus aureus, and untyped pneumococci were uncovered in several sputum cultures. This case exemplifies the infectious type of bronchial asthma, most liable to respond to nebulization.

Case 4—A white female adult was admitted to the hospital with slight frontal headache, cough, muco-purulent sputum and malaise of seven weeks' duration. On occasion there were night sweats and a feeling of feverishness. A chest survey, including roentgenograms, failed to identify any pulmonary lesion. There was a mixed flora of bacteria in several sputum specimens. She had a mild nasopharyngitis and bilateral maxillary sinusitis, confirmed by x-rays. However, no pus was obtained by antral puncture. There were occasional inconstant low-pitched musical rales and medium inspiratory rales bilaterally. Prior to hospitalization, nose and throat treatment, oral sulfonamides, expectorants and general treatment were prescribed, but the results were poor. On the fifth day of nebulization therapy there was an absence of all symptoms and abnormal physical signs. This is an example of bronchitis secondary to upper respiratory infection, running a subacute to chronic course, with an excellent response.

Case 5—A white male adult, employee of the Panama Canal, was admitted with a bronchopneumonia of the right lower lobe. There was a mixed flora of organisms in the sputum. The parenchymal lesion cleared on a conservative regime within seven days but the patient continued to cough and expectorate muco-purulent sputum. The x-ray at this time showed accentuated pulmonary markings and there were occasional low-pitched musical rales, indicative of a post-pneumonic bronchitis. This condition was resistent to all forms of treatment, including iodides, expectorants, sedation and oral sulfonamides. As the signs did not abate after several weeks, he was transferred to the chest service, where sulfa nebulization was initiated. Within two days he showed moderate improvement and at the end of five days he was completely symptom-free with no clinical evidence of a respiratory infection.

COMMENT

The effect of the direct application of sulfonamides to mucous membranes has been the subject of comment and study. Fletcher[8] reported the caustic action of the drug on mucosal tissue. Hunnicutt,[9] employing a 5 per cent solution of sodium sulfathiazole in an experimental study on the mouse, noted an initial irritation of the nasal mucosa with a subsidence of the reaction and no harmful end result. In this series there were two cases of mild local reaction. There was a rapid dilution of the original solution with a subsequent decrease in alkalinity as a result of the buffering action of the respiratory secretions, so that the chance of an irritating effect was minimized.

The efficiency of this form of therapy is based on the intimate contact of the sulfonamides with the respiratory mucosa harboring pathogenic organisms. The solution of sodium sulfathiazole in contact with body fluids precipitates out as sulfathiazole and its bacteriostatic property produces a salutary effect. Although oral administration of the drug should produce similar results, local concentration by the inhalation method probably accounts for the disparity in therapeutic response as observed in several instances. The advantages of localization of drug treatment for concentration purposes and the avoidance of systemic reactions are noteworthy benefits.

It is interesting to speculate on the various modifications of this method. The selection of the drug need not be confined to sulfathiazole, as other sulfa compounds may be employed, depending upon the susceptibility of the organisms involved. An aqueous solution of sulfathiazole with desoxyephedrine hydrochloride,[10] which has been employed in the form of packs for sinusitis, laryngitis and tracheitis, is worthy of trial in certain constrictive types of bronchitis and bronchial asthma. As far as the vehicle is concerned, there are also other possibilities. Oxygen may be replaced by a mixture of 10 per cent carbon dioxide and 90 per cent oxygen, as suggested by Banyai and Cadden,[11] in cases of thick and tenacious sputum creating difficult expectoration. Barach[12] has recommended the use of an oxygen-helium mixture, which may be substituted for oxygen in cases of status asthmaticus. Sulfa nebulization may also be valuable when it is combined with positive-pressure oxygen[13] for serious respiratory disorders and pulmonary edema, where secondary infection is a factor.

The procedure, as expected, failed completely in bronchial asthma due to non-bacterial inhalants. Cases of chronic bronchitis associated with pulmonary emphysema and bronchiectasis, both irreversible processes, may be favorably influenced but the effects

are not curative. Such lesions as sinusitis with secondary bronchitis, subacute or chronic bronchitis secondary to other upper respiratory infections, post-pneumonic bronchitis and infectious asthmatoid bronchitis often improve remarkably and the results may be curative. Based on an analysis of the various clinical entities, it was generally noted that chronic bronchial lesions of bacterial origin benefited by a course of sulfa nebulization.

SUMMARY

1) A group of 50 patients with diverse infectious bronchial lesions, refractory to other forms of therapy, was treated by the inhalation of nebulized solution (5%) of sodium sulfathiazole.

2) The method of study and of the procedure was described.

3) In 43 cases or 86 per cent of the series there was a definite improvement.

4) Several illustrative case reports were presented.

5) Modifications of the procedure were suggested for further clinical trials.

6) This preliminary survey indicates the promising therapeutic possibility of sulfa nebulization in a resistant type of respiratory infection.

RESUMEN

1) Un grupo de 50 pacientes con diferentes lesiones bronquiales infecciosas, refractarias a otros tratamientos, fueron tratados con la inhalación de nébulas de una solución (5%) de sulfatiazol sódico.

2) Se describe el método de investigación y el procedimiento usado.

3) Hubo mejoría bien definida en 43 casos, o sea en el 86 por ciento de esta serie.

4) Se presenta informes de varios casos ilustrativos.

5) Se sugiere modificaciones del procedimiento que pueden emplearse en ensayos clínicos adicionales.

6) Este examen preliminar indica las prometedoras posibilidades terapéuticas de nébulas de sulfa en un tipo resistente de infección respiratoria.

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Funnel Chest: Report of Case Successfully Treated by Chondro-sternal Resection

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Funnel chest (chone-chondrosternon, pectus excavatum) usually is a congenital, but may be an acquired, condition. There is a depression of the lower portion of the sternum with the costal cartilages, which has a tendency to become worse as the individual grows older. Chone-chondrosternon is the name that has been given to the condition by Ochsner and DeBakey,[1] who have made a very extensive review of the literature and reported a case of their own. They very carefully reviewed the records of thirty-two patients who had been operated upon during the past twenty-seven years. Since then, Brown[2] reports on three radically operated cases and reports four cases developing pectus excavatum upon whom a palliative operation was performed. Haberlin[3] has also successfully operated upon one case. The very fact that so few cases have been operated upon does not mean that the condition does not occur more frequently with symptoms severe enough to require operation.

Operation undoubtedly has been denied many patients who could have been benefited surgically. There are three methods of attacking such a condition. The three types of operative procedure are: (1) Chondro-sternal resection. Ten cases had been treated by this procedure with successful results in eight, and death occurred in two. (2) T-Sternotomy, with or without costal-cartilage division, was carried out in fourteen cases. Eight cases were successful, two failed, and death resulted in four. (3) Sternal mobilization with chondral division or resection. This undoubtedly is the operation of choice, because a better thoracic cage will result. Eight cases have been operated upon by this procedure with seven satisfactory results and one failure.

I wish to add a case in which chondro-sternal resection was carried out. Because of the marked deformity of the sternum with rotation, it was felt that resection of the sternum and cartilages was the operation of choice; and a very satisfactory end result was obtained.

Case Report—The patient is a white female, age 21, whose chief complaints were shortness of breath, increasing deformity of the chest and pain and discomfort in the chest. Some deformity had been present as long as she could remember. It had increased in the past two years. She had been working in a defense plant up until the present time. She found

it necessary to stop work because of increasing difficulty with shortness of breath and pain in her chest, and a feeling of pressure on the heart. The pulse rate had been increasing. She had a feeling of a constriction of the chest and she was quite conscious of her heart. These symptoms had become much worse within the last three or four months and in addition to her symptoms she had become quite sensitive about the deformity of the chest and a tendency to stooping of her shoulders. There was also some lack in development of the left breast, it being about half the size of the right breast. Her general physical examination otherwise was negative. There were no abnormal heart sounds. The x-ray of the chest revealed some displacement of the heart toward the right side. The costal arch, on the left, shingled over the sternum so that about one-half of the sternum was under the arch, and there was a marked depression of the sternum with the costal arch on the right side and to the lesser degree on the left side. The blood pressure was 120 over 70, the pulse was 100, and there was a rather marked tendency for the patient to bend forward from the shoulders. The patient had no history of injury, and her general physical condition had always been good. There was no history in the family of abnormality in development as far as she could determine. The urine analysis, blood counts and blood Wassermann tests were all within normal limits.

She was operated upon January 10, 1944. A resection of the xiphoid and body of the sternum, together with the 3rd, 4th, 5th and 6th chondral cartilages on each side was carried out through a curved incision over the sternum. The chondral cartilages on the left were abnormally attached to about the level of the 3rd chondral articulation. They all seemed to run up to this area in a knob formation. They were shingled over each other and the sternum was shingled under these cartilages on the left

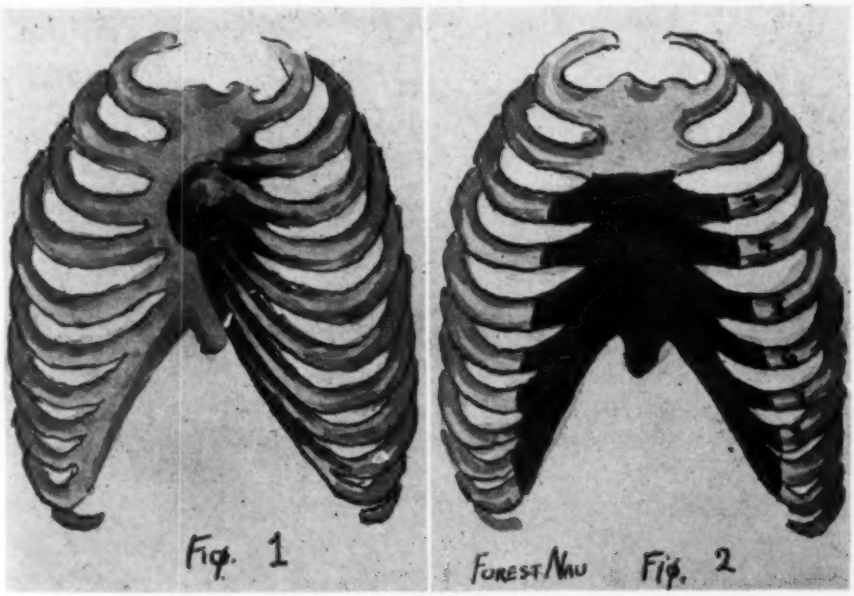
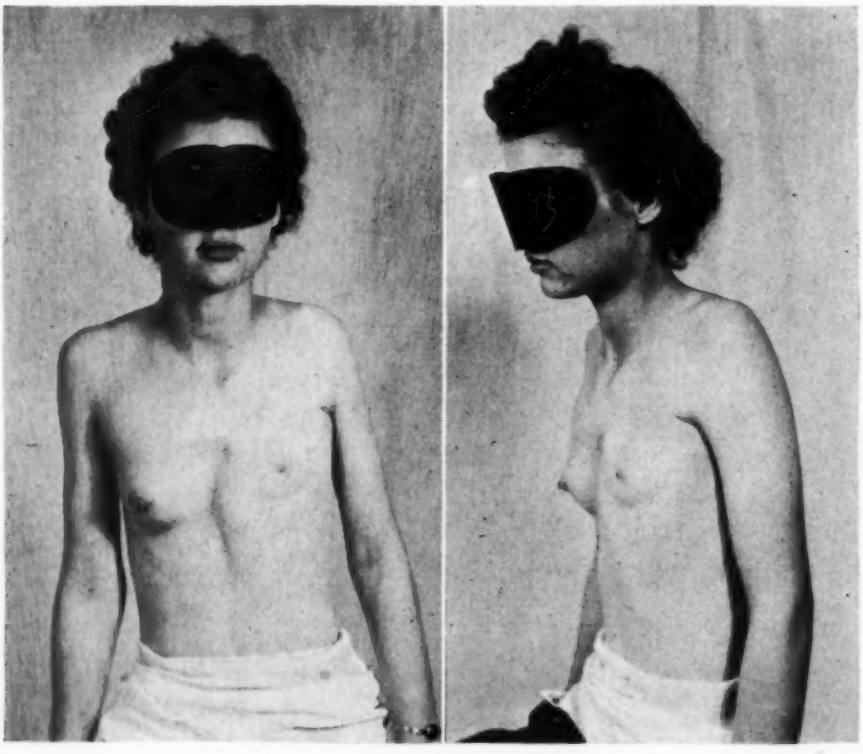


Fig. 1—Artist's drawing of shingling of the ribs over the sternum. *Fig. 2*—Showing the amount of chondrosternal resection that was carried out.

side. The chondral arch was completely resected with the cartilages and the pericardium peeled away from the posterior part of the sternum. The pleura and pericardium were dissected away without entering into either of these cavities. Due to the marked deformity over the body of the sternum I felt it advisable to resect the sternum rather than to fracture it and hold it in place with wire. The bleeding from the body of the sternum was controlled by bone wax. One penrose drain was left over the pericardium and brought out through the lower angle of the incision. Five grams of sulfanilamide was left in the wound. The patient was given 500 cc. of blood at the completion of the operation. The operation was done under intratracheal cyclopropane anesthesia. The whole procedure took about an hour and forty minutes and really worked out very well. The patient's immediate postoperative course was very good. She showed no evidence of any cardiac embarrassment, or abnormality. Her temperature never ran over 100°; on the fifth day she was allowed to sit on the side of the bed, and by the end of the week she was out of bed and walking around in the ward. She was allowed to leave the hospital in ten days and has progressively improved. The heart action has been perfectly normal, with rather active pulsation over the precardial area, but this in no way disturbs the patient. The deformity has been largely corrected. She does not have the feeling of pulling over of her chest, and two months after operation she was allowed to return to her usual duties in a defense plant. Her convalescence really was without incident.



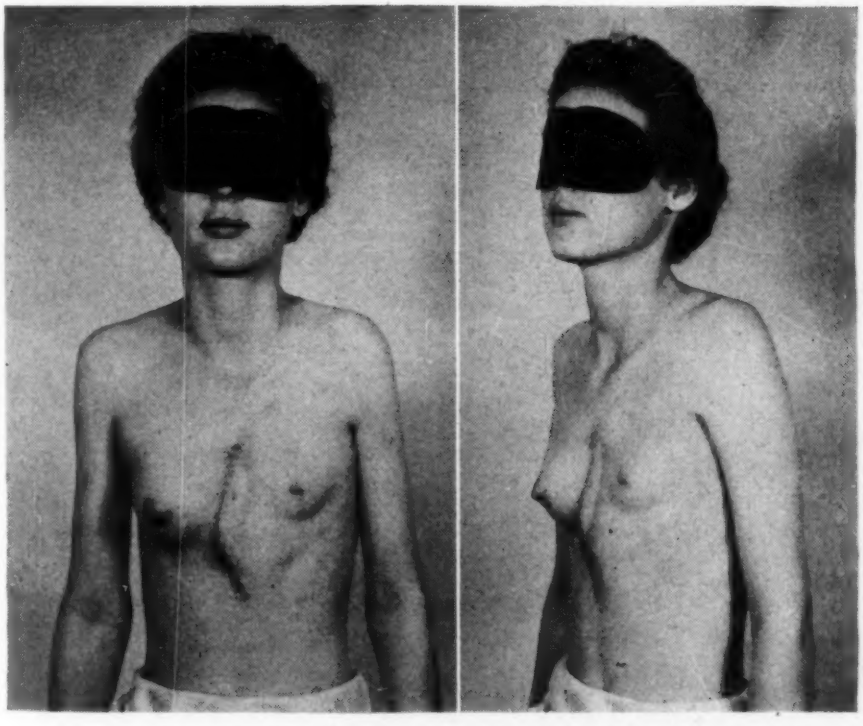
Front and side views preoperatively.

SUMMARY

Another case of successful resection of the sternum with the chondral cartilages on each side for depression of the sternum and costal cartilages has been reported. It is felt that the operation of choice in these cases would be sternal mobilization rather than resection, but in this case, due to the marked deformity of the costal arch, together with the rotation of the sternum, resection was necessary. It is true the entire literature does not contain enough case reports to actively determine which procedure will carry the highest incidence of cure. The mortality rate in resection of the sternum should be no higher than the mortality rate in sternal mobilization. This brings the number of cases that have been operated upon by a radical procedure reported in the literature to thirty-eight. In children a less radical procedure of dividing the diaphragmatic attachment to the sternum will usually stop the progress of the condition.

RESUMEN

Se informa sobre otro caso de resección del esternón y de los cartílagos de ambos lados, ejecutada con buen éxito para corregir la depresión del esternón y de los cartílagos costales. Se opina que



Front and side views two weeks postoperatively.

la operación de elección en estos casos sería la movilización del esternón más bien que la resección; pero en este caso, debido a la gran deformidad del arco costal y a la rotación del esternón, la resección fue necesaria. Es cierto que la entera literatura no contiene suficiente número de informes de casos para determinar adecuadamente cuál procedimiento obtendría el mayor número de curaciones. La mortalidad en la resección del esternón no debe ser más alta que la mortalidad en la movilización del esternón. Este eleva a treinta y ocho el número de casos sometidos a una operación radical, que se han presentado en la literatura. En los niños el procedimiento menos radical de dividir la unión del diafragma con el esternón por lo general interrumpe el progreso de este estado.

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Broncholithiasis

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Although this condition was first fully described by Schenck in 1600, van Ordstrand, Moore & Harris state that only 26 cases of broncholithiasis—as distinct from various types of intra pulmonary calcifications—have been reported in the English literature up to the end of 1941.[1] They would appear, therefore, to be comparatively rare. They may be primarily of endobronchial formation but much more commonly originate outside the bronchus and ultimately erode through the wall. Most frequently they represent the end stage of a primary tuberculous lesion in the tracheobronchial glands,[2] but may result from other types of pulmonary inflammation and suppuration.

In the large majority of reported cases the diagnosis has been based on expectorated stones and in a few on post mortem findings. In later years the bronchoscope has played an increasingly important role both in diagnosis and in treatment. Differentiation can usually be made by this means between a broncholith and the condition with which it is most likely to be confused—a bronchogenic carcinoma. X-rays will, with certainty, demonstrate only the fact of obstruction or partial obstruction of a bronchus.

The physical nature of bronchial concretions varies considerably. They may be hard and firm and, generally, irregular in outline, or of a putty-like consistency; and with the former type considerable care must be used and only gentle and careful manipulations are permissible in attempts to dislodge them through the bronchoscope as pneumothoraces and fatal hemorrhages have followed such maneuvers. It is interesting to speculate why calcium should be deposited in the lung and its lymphatic system so much more frequently than in other portions of the body, and, as has been suggested by Wells,[4] it seems probable that this is due to the greater solubility of calcium salts in weakly acid media than in neutral or alkaline ones. Thus the removal of a large part of the CO_2 from the blood in the lungs would tend to cause some precipitation of lime. Superficially it might seem that an increased consumption of milk and vitamin D would tend to increase the formation of calcium concretions; but, it is to be remembered that the saturation of a solution with a salt such as calcium phosphate depends not on the

total quantities of calcium and phosphorous present but on the product of the concentrations of Ca and PO_4 ions ($Ca^{++} \times PO_4^{---}$) in the solution; and in general it will be found that the deposition of lime in devitalized tissue depends on the fact that CO_2 production is minimal in such tissues.

The symptoms are those chiefly of any bronchial obstruction, the most prominent being cough which is frequently of a wheezing, spasmodic, asthmatic type and often accompanied by pain in the parasternal region of the chest. This pain is apt to be sharp and boring in character during the spasms of coughing and rather dull between them, and in and by itself is suggestive of lithiasis. Varying degrees of hemoptysis from streaking to frank and copious hemorrhages are commonly present. Since both bronchiectasis and lung abscess[3] may be associated phenomena, periodic purulent sputum intervening in a generally mucoid or muco-hemorrhagic expectoration may sometimes be seen. Attacks of dyspnoea may be striking, especially prior to the expectoration of a stone, and relief following its expulsion may be equally marked. Weight loss is not usually great, though loss of sleep through pain and anxiety may lead to some. Most typically there is periodic loss and gain corresponding to the cycles of pain and ease.

The following is a report of a case which came under our care in January, 1942:

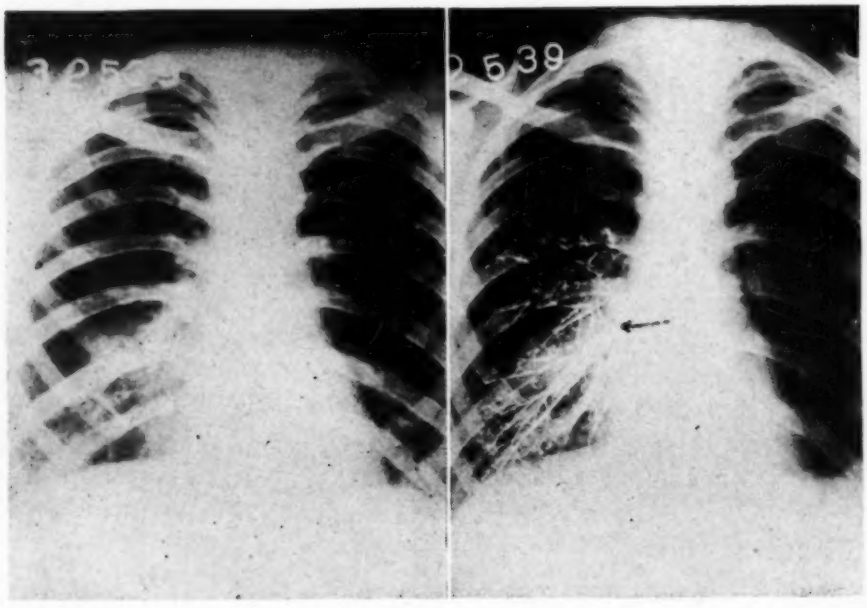

Fig. 1 *Fig. 2*

Fig. 1—Atelectasis of middle lobe. *Fig. 2*—Lipiodol injection showing point of block.

Mrs. E. H., age 33 years, was admitted to the Royal Alexandra Hospital on January 24, 1942. Her complaint was of cough for 12 months, recurrent hemoptysis for 10 months and periodic pain to the right and left of mid sternum. In the previous October she had been in another hospital because of copious hemorrhages which necessitated transfusions. These episodes of bleeding were always immediately preceded by sharp, intense, needle-like pains in the anterior central portion of the chest. A flat film at that time showed atelectasis of the right middle lobe (Fig. 1). A film taken after lipiodol injection, however, failed to demonstrate any bronchial obstruction and a bronchoscopic biopsy was reported as consisting of chronic inflammatory tissue. She had lost some weight during the past summer but had subsequently regained most of it. Her cough was dry and unproductive, but later the sputum was somewhat freer, mucoid and blood-tinged, and on one occasion appeared to consist of pure pus. The temperature at the time of admission was 98.4°, pulse 80 and respirations 20. The highest recorded temperature up to the time of operation was 99.2°. At no time did the sputum show tubercle bacilli or fungi, and no tumor cells were found.

On January 27, bronchoscopy was carried out by Dr. J. G. Young, and the findings were a normal trachea and right bronchial tree. X-ray following lipiodol injection showed an obstruction of the right middle lobe bronchus (Figs. 2, 3 and 4). On February 16, bronchoscopy was repeated and again a normal tree, bronchoscopically, was reported. Actually, as was later demonstrated, the swelling proximal to the obstruction coming at the curve of the bronchus prevented vision beyond it, and gave the appearance of normal mucosa.

Because of the clearly demonstrated bronchial block and the distal atelectasis and the continued bleeding, a bronchogenic carcinoma appeared to be the most likely diagnosis, and operation was decided upon.

On February 26, a preliminary and satisfactory artificial pneumothorax was induced, and on March 2, lobectomy was performed (W. S. A.).

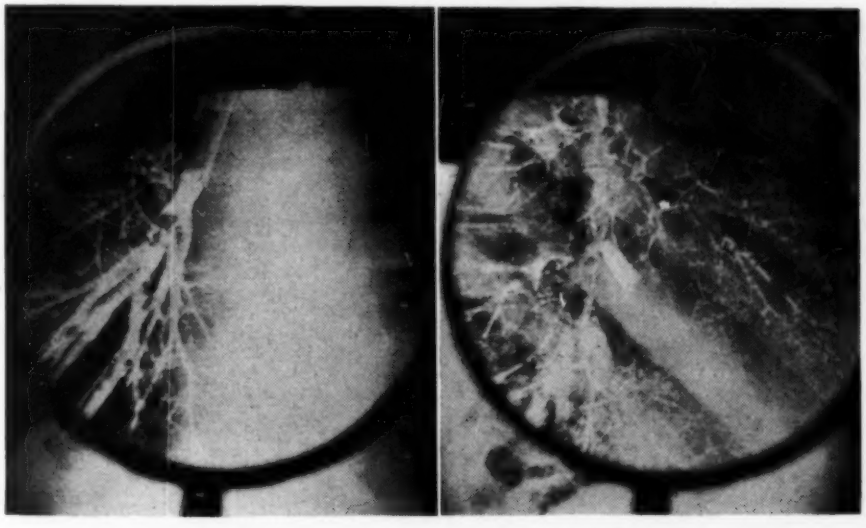

Fig. 3 *Fig. 4*

Fig. 3—Detail of bronchi shown in Fig. 2. *Fig. 4*—Right oblique. Some of the lipiodol has been coughed up.

Under cyclopropane endotracheal anaesthesia with the patient lying on her left side, the right pleural cavity was opened through the sixth interspace and the sixth and seventh ribs fractured posteriorly to increase the exposure. There were only a few adhesions in the region of the diaphragm, otherwise the lung was lying free. The pleura over the middle lobe was covered with fibrin; it had obviously been collapsed for some time and was the seat of an inflammatory process. The upper and lower lobes were uniformly crepitant throughout. No growth could be palpated anywhere and it was decided that the obstruction demonstrated radiologically and producing the atelectatic middle lobe was probably due to a small benign adenoma. Hence, instead of the contemplated radical pneumonectomy, a middle lobe dissection lobectomy was carried out. Each vessel and the main middle lobe bronchus were dissected out and disposed of separately. On dividing the bronchus, a rough hard broncholith about 1 cm. in diameter was seen to be lying in an ulcerated area just distal to the level of section.

Unfortunately, in ligating the structures at the root of the middle lobe and repairing the defect, it was felt that the main bronchus to the lower lobe had been kinked and that partial obstruction might result. For this reason, the lower lobe was also removed. The final stump, with all structures ligated separately, was dusted with sulfathiazole crystals and then covered with the sleeve of pleura that had purposely been left.

The chest cavity was closed in the usual manner and closed drainage instituted through a catheter in the 9th space posterolaterally, leading down to a drainage bottle with a water seal. The trachea and upper bronchi were thoroughly aspirated. The patient received 1100 cc. of citrated blood and plasma during the operation, which was tolerated well.

Postoperatively, there was considerable drainage from the pleural cavity, but the catheter finally plugged and it was necessary after the first week to aspirate the chest occasionally to relieve dyspnoea. During the third week her temperature rose to 104° but after a severe paroxysm

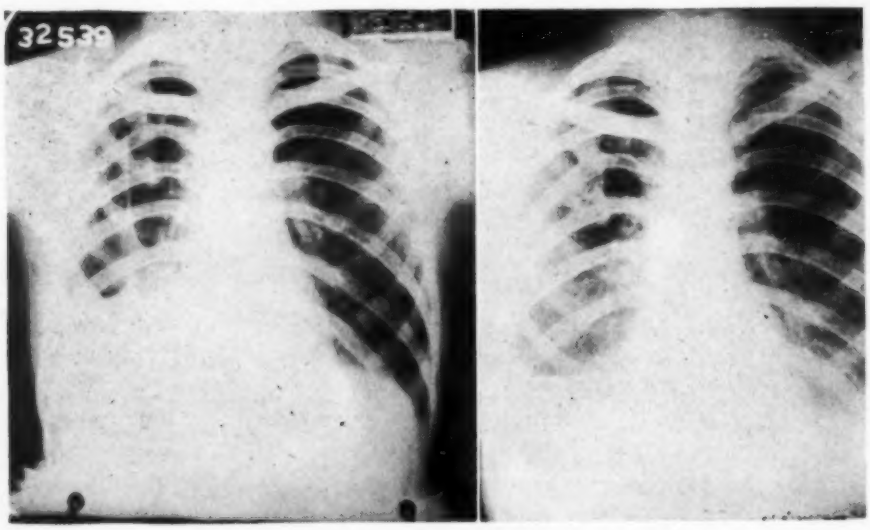

Fig. 5 *Fig. 6*

Fig. 5—April, 1942. Partial expansion of upper lobe. *Fig. 6*—Complete expansion of upper lobe. Bony bridge between cut ribs.

of coughing when she brought up an ounce or so of thick blood-stained pus, her temperature came down to normal, and it was apparent that an abscess in the stump had fortunately ruptured into the bronchial tree and drained.

However, the abscess cavity took a long time to heal, and when it was not emptying freely the temperature would shoot up to 102°. With postural drainage over a 3-week period, her sputum decreased from 8 ounces daily to nil.

She was discharged on the 48th postoperative day (April 18) after being afebrile for a week with a small amount of fluid and considerable air still in the chest cavity (Fig. 5). There was no cough, she felt well and was gaining weight.

On May 6, 1942, she reported that the previous day she had done all the ironing for a family of seven, as well as the cooking and general housework. She had practically no cough except in a cold wind; tended to tire rather easily but felt well and had put on 7 pounds in weight. Lung was still only partially expanded and there was a thickened edge of pleura running from diaphragm to apex.

A re-check on October 27, 1943, shows that she has gained a total of 30 pounds in weight, looks well and feels well. The upper lobe now completely fills the chest cavity, and has a normal appearance (Fig. 6). There is a bridge of calcium joining two ribs where they were cut.

PATHOLOGICAL REPORT

Specimen consists of right lower and middle lobes. The lower lobe throughout is collapsed but seems to be normal. The inferior portion middle lobe is comparatively normal. The small bronchi supplying this area contain clumps of muco-purulent exudate. Three calculi were found in bronchi supplying upper two-thirds of this lobe. The largest concretion, measuring 0.8 cms. in diameter, was lying in a dilated area of the main bronchus surrounded with blood and on the posterior surface of this cavity was a vessel, the wall of which was perforated. This upper two-thirds of the middle lobe on section shows chronic atelectasis with abscesses of bronchi, partial destruction of bronchial walls and peri-bronchial purulent infiltration. The collapsed air cells contain pus cells and there is considerable scarring of lung tissue.

Pathologic Diagnosis—Chronic atelectasis of middle lobe of right lung with chronic abscess formation; chronic suppurative bronchitis with multiple bronchial calculi.

These concretions had originated in a calcified lymphatic gland lying behind the bronchus and had perforated through it, eroding a blood vessel in the process.

SUMMARY

Broncholithiasis is a comparatively rare condition. The calculi may be primarily of endobronchial formation, but much more commonly originate outside the bronchus and ultimately erode through the wall. Most frequently they represent the end stage of

a primary tuberculous lesion in the tracheobronchial glands.

In the large majority of reported cases the diagnosis has been based on expectorated stones and in a few on post mortem findings. In later years the bronchoscope has played an increasingly important role both in diagnosis and in treatment.

The symptoms are chiefly those of any bronchial obstruction, the most prominent being cough which is frequently of a wheezing, spasmodic, asthmatic type and often accompanied by parasternal pain. Varying degrees of hemoptysis from streaking to frank and copious hemorrhages are commonly present.

A case of broncholithiasis is reported.

RESUMEN

La broncolitiasis es un estado comparativamente raro. Los cálculos pueden ser de origen endobronquial, pero con mucha más frecuencia se originan afuera del bronquio y finalmente atraviesan la pared bronquial. Con mayor frecuencia representan el período final de una lesión tuberculosa primaria en los ganglios tráqueobronquiales.

En la gran mayoría de los casos presentados se ha basado el diagnóstico en piedras expectoradas y, en unos cuantos, en hallazgos autópsicos. En años recientes, el broncoscopio ha desempeñado un papel cada vez más importante tanto en el diagnóstico como en el tratamiento.

Los síntomas son principalmente los de cualquiera obstrucción bronquial, siendo el más conspícuo la tos, que con frecuencia es ruidosa, espasmódica, de tipo asmático, y a menudo acompañada de dolor paraesternal. Sobreviene comúnmente hemoptisis de varios grados, desde esputo veteado con sangre hasta hemorragias sueltas y abundantes.

Se informa sobre un caso de broncolitiasis.

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Cystic Disease of the Lung With Iodized Oil Studies

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Cystic disease of the lung has, in recent years, been assuming more and more importance in diagnosis, as evidenced by the number of papers on this subject which have appeared in medical literature. Our knowledge of this condition was meager until 1925, although it had been mentioned in the literature much earlier. The first case report is variously credited to Fontanus in 1638, Bartholinus in 1689 and to Meyers in 1858.

In 1925 A. R. Koontz[3] published the first comprehensive report. At this time he had collected 108 cases and added one of his own. In 1935 Pearson[4] reported a total of 172 cases. Schenck[7] in 1937 stated that 381 cases had been reported.

Koontz and Pearson both draw attention to the fact that this condition has been reported under varying titles. These include acquired bronchiectasis, atelectatic bronchiectasis, bronchiectatic atelectasis, fetal bronchiectasis, agenetic bronchiectasis, localized emphysema, pneumothorax, saccular degeneration of the lung, congenital cystic malformation of the lung, honeycomb lung, and pneumatocele.

There has been much discussion by various writers as to whether cystic disease of the lung is congenital or acquired. Pollock and Marvin[6] state: "Congenital cystic disease of the lung we believe to be a definite disease entity and one of extremely rare occurrence. The cystic condition of the lung is secondary to a narrowing of the main bronchus or bronchi of the involved lobe or lobes and it is this narrowing which is congenital."

King and Harris[2] put forth a unique theory for formation of these cysts. They quote S. S. Simpkins as saying that the bronchi develop as small ramifications of entodermal tissue which become canalized almost immediately. King and Harris think that an unknown process interferes with canalization at some point proximal to the termination of a particular ramification, resulting in an occlusion at that point. Beyond the occlusion, canalization begins again, and thus an isolated canalized segment is formed. The mucous mem-

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brane of this segment in turn assumes a normal secretory function, and a cyst is formed.

Pierce[5] in a recent article considers most cases to be acquired and only a few to be of true congenital origin. This author further points out that in the acquired type, bronchitis to a minor degree and bronchopneumonia to a major degree are the causative factors in the production of these so-called "cystic" changes in the lung. Pierce further offers the following classification of acquired lesions: (1) Cystic bronchiectasis. (2) Chronic interstitial pneumonitis with emphysema. (3) Chronic bullous emphysema, and (4) Pneumatocele (localized lobular ectasia).

The morbid anatomy is clearly epitomized by Koontz.[3] He recognizes two general types. Those of the first type are bronchial dilatations as shown by persisting muscle fibers and cartilage in the walls. The second group are cavities resembling emphysematous blebs lying subpleurally. There are of course all degrees of gradation and transition types between these two.

The epithelial lining is usually columnar and ciliated. It may be devoid of cilia, cuboidal or flat or the membrana propria may be denuded. Mucous glands are at times present and may form retention cysts.

Most authorities state that a complete lack of pigment in congenital lesions distinguishes them from the acquired variety. The following salient features have been pointed out about cysts. They may be single or multiple, unilocular or multilocular. They may vary in size from minute blebs to enormous cysts that occupy half or more of a thoracic cavity. Either fluid or air or both may be contained in a cystic space. The shape may be spherical or ovoid. The pulmonary alveoli in the vicinity of the cysts may be collapsed or atelectatic or may have failed to develop. This condition may occur in any portion of one or both lungs. Male or female may be affected equally often.

From the reports it would seem that complications and concurrent affections occur less often than one might expect. Cases are recorded in which cysts were associated with hydrothorax, pneumothorax, empyema, bronchopneumonia, lobar pneumonia and, rarely, tuberculosis. In one recorded case it coexisted with sarcoma.

The more common symptoms of cystic disease are dyspnea, cyanosis, cough, cardiac palpitation and rarely hemoptysis.

Wood[8] pointed out that symptoms vary with the extent of lesion, site, and presence or absence of increased intrathoracic pressure. He thinks that the presence of cysts should always be considered in infants who have recurring attacks of dyspnea and cyanosis; and in adults with progressive dyspnea without other known cause.

The main condition to be considered in differential diagnosis is spontaneous pneumothorax. The significant points of differentiation are: (1) There is no history of sudden onset; (2) Subjective symptoms are normal with the exception of shortness of breath in far-advanced cases; (3) No pathological change can be proven as an etiological factor of spontaneous pneumothorax; (4) X-ray and physical findings remain unchanged over long periods of time; (5) The condition is often accidentally discovered; (6) X-ray findings differ from those of spontaneous pneumothorax in the following respects: (a) There is no displacement of the mediastinum or of the mediastinal organs; (b) The compressed lobe cannot be seen; (c) The fine strands traversing the space are in wide sweeping curves and not straight as one would expect to find the small taut adhesions traversing a pneumothorax space.

The roentgenologic features of pulmonary cystic disease and the ease with which they can be diagnosed vary according to the con-

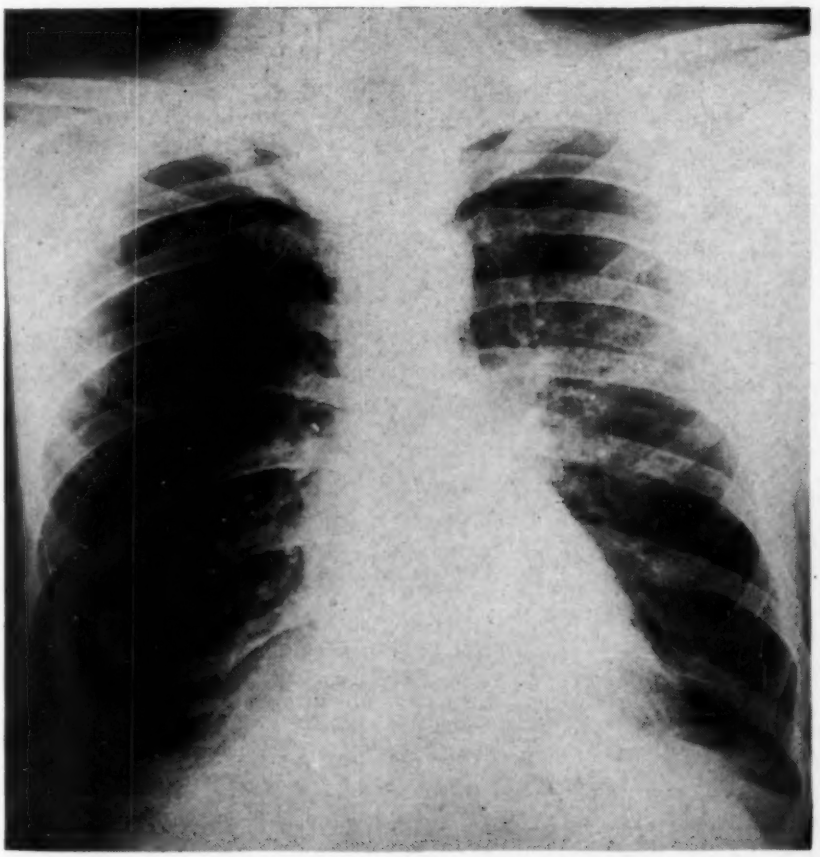

Fig. 1—P. A. x-ray of chest. Note the disease in the right lung as shown by the fine strands traversing the space in wide sweeping curves. Disease in the left upper is also seen as fine strands, shorter than on the opposite side.

tent, size, number and situation of the cysts and the presence or absence of complications or concurrent disease.

Cysts filled with fluid are not easily distinguished from several other pulmonary conditions. This type of cyst without an inflammatory zone around it is usually round or ovoid and casts a uniformly dense and sharply circumscribed shadows. They are most often single, relatively large and resemble benign neoplasm, primary or secondary neoplasm, primary or secondary metastatic new growth, hydatid cyst, dermoid cyst or aneurysm. Even after careful study it may be necessary to make a nonspecific diagnosis of tumor of the lung. An infected fluid-filled cyst surrounded by an irregular zone of reactive inflammation resembles and in most cases will be mistaken for an abscess or pneumonic consolidation.

Cysts containing both air and fluid must be differentiated from abscess, tuberculous cavitation and draining hydatid cyst.

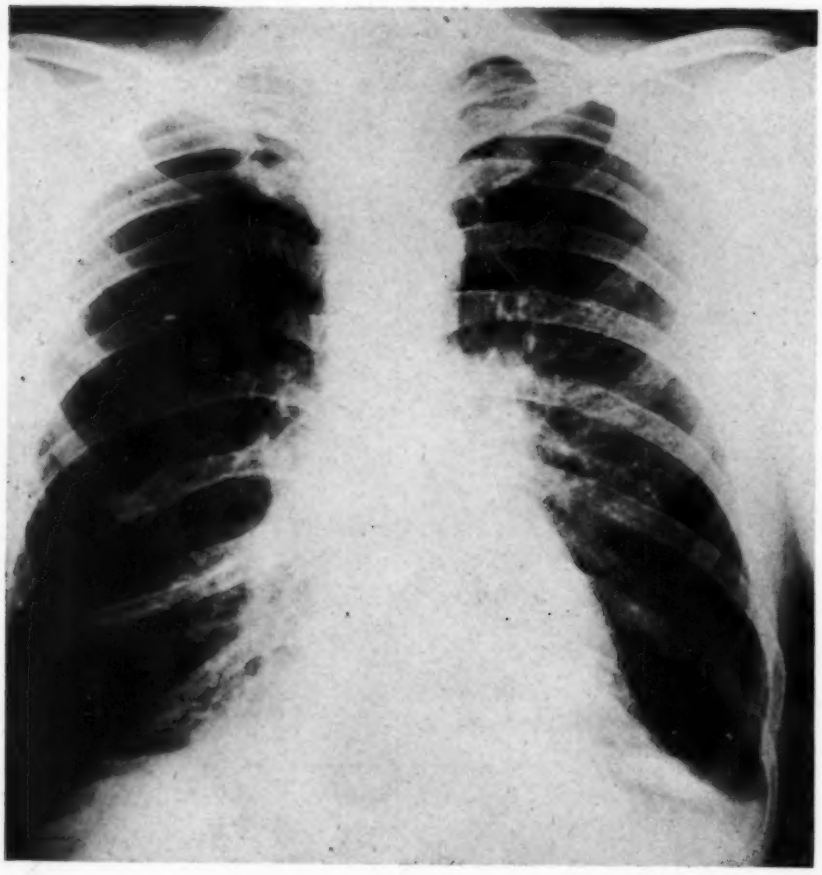

Fig. 2—P. A. x-ray of chest following induction of artificial pneumothorax on the right side. Only a small pocket of air is seen laterally at the base. *(Note*: This film was retouched slightly to bring out the margin of this space.) The wall of the cyst is seen bulging into the pleural air space.

Large air-filled cysts can be distinguished on the x-ray with a high degree of accuracy. They usually are single or not exceeding two or three in number. There is a radioparent area devoid of normal pulmonary markings. The part of the wall of the cyst in contact with the lung appears as a regularly curved line or lines. There are cases in which it is difficult to differentiate. In these cases the following diagnostic procedures may be employed: (1) Wilson's procedure of inducing slight artificial pneumothorax, which demonstrates the lateral lung margin and establishes the fact that one is not dealing with a spontaneous pneumothorax; (2) Thoracentesis may be performed, with needle puncture of the structureless space and injection of 1 to 2 cc. of iodized oil into it; (3) An artificial pneumothorax may be established and 10 to 15 cc. of iodized oil injected into the pleural space. The patient is then manipulated around to allow the oil to spread out and come in contact with all the pleural surfaces. X-rays immediately following this are very distinctive. We used this procedure in our case. From a study of the literature we believe this to be the first time that this procedure has been used for diagnosis of cystic disease. (4) Iodized oil bronchograms may at times be helpful.

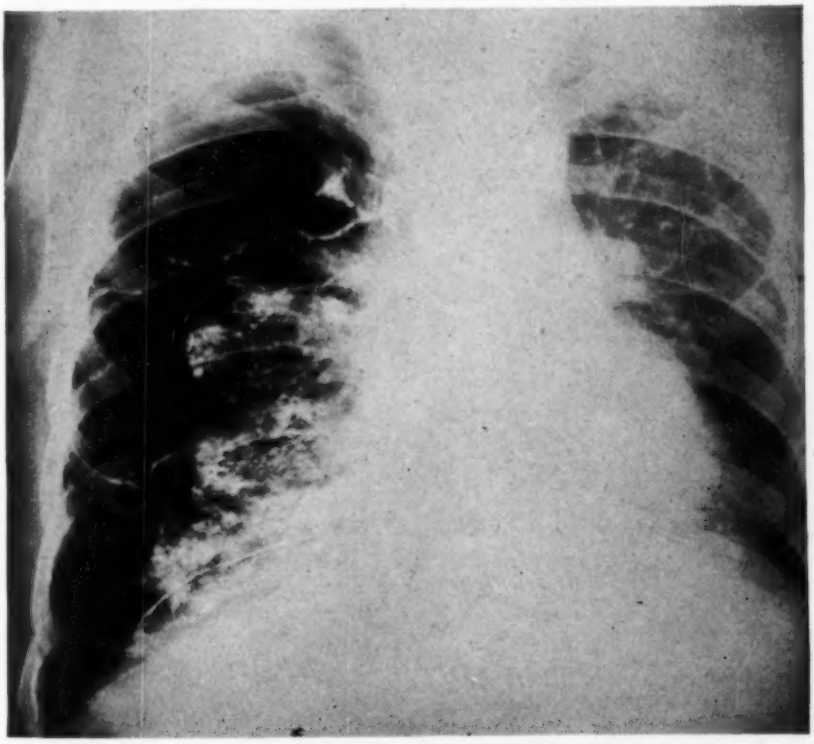

Fig. 3—A. P. x-ray of chest in prone position following injection of iodized oil into the pleural space. The walls of the cyst are now clearly seen and it is evident that there are two separate spaces—cystic and pleural.

Multiple, grouped air-filled cysts are not uncommon. They appear as delicate, complete or incomplete rings, or as a complex network of shadows resembling cobwebs. These must be distinguished from emphysema, diaphragmatic hernia, and bronchiectasis.

Cystic disease of the lung may be complicated by pneumothorax from rupture of a cyst, hydrothorax, empyema, pneumonia, tuberculosis, carcinoma, or any of the other diseases that may attack the lung. These may make the diagnosis very difficult or impossible.

Final diagnosis takes complete cooperation between the clinician and roentgenologist. Correlation of roentgenological and clinical data is essential.

Treatment in general is unsatisfactory. Cole and Nalls[1] say that treatment is dependent on the size, number, location and type of cysts, the presence or absence of infection, and the urgency for relief of symptoms. Wood[8] states that some cases are improved

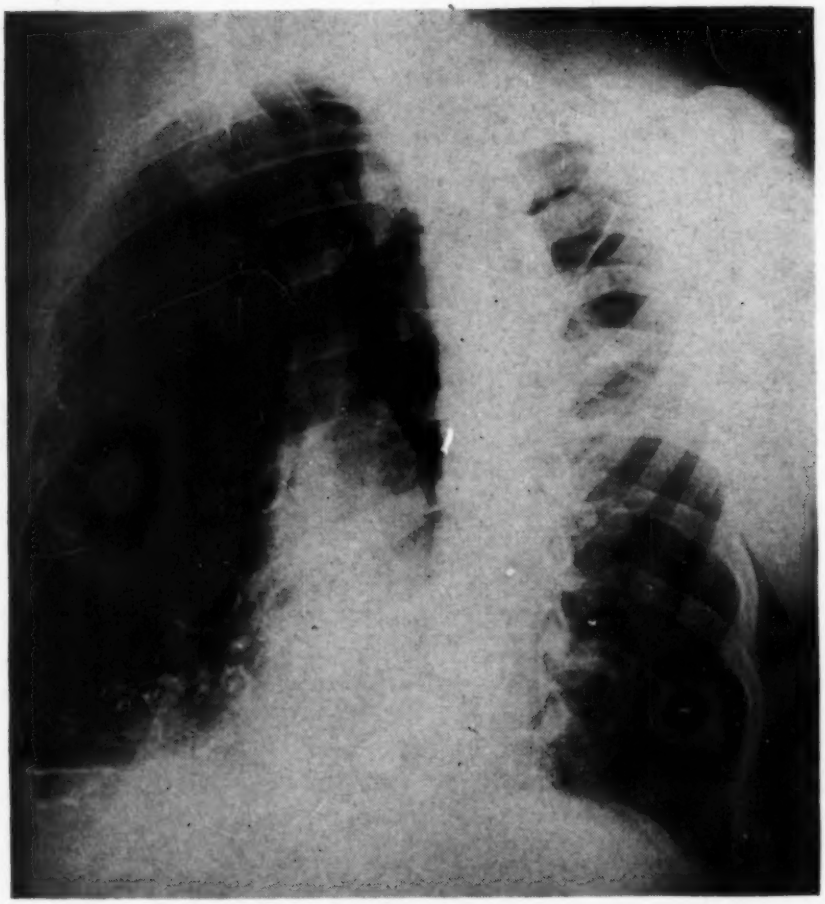

Fig. 4—X-ray of chest in oblique position following injection of iodized oil into the pleural space, showing findings similar to Fig. 3.

following bronchoscopic aspiration and injection of lipiodol; but he also thinks that treatment should be limited to infected cysts which are amenable to this type of drainage. Wood reports two cases of fluid-containing cysts which were removed surgically. Schenck[7] advocates extirpation of the cysts, saying that at times lobectomy should be resorted to, but adds that the procedure is attended with grave danger. Naturally, if enough lung units are destroyed, there is no surgical or medical procedure which will benefit the patient.

CASE REPORT

G. D., an adult white male, aged 42; occupation, bus driver. Family history not significant. Previous personal history: Patient never was as husky as the other children in his grade at school. Had influenza in 1918. Otherwise comparatively well until present illness.

Present Illness—Patient had recurring attacks of shortness of breath, especially in the morning on arising, during May, 1943. In June he had what was diagnosed as "pneumonia"—symptoms actually were only some increase in shortness of breath together with coughing. X-rays taken in July were read as showing "upper right lung collapsed and lower right full of pneumonia—no tuberculosis." X-rays in August showed "pneumonia clearing some, right upper still collapsed and left upper collapsing some." A chest specialist was then consulted. Sputum studies were made for tubercle bacilli and one sputum was reported positive, Gaffky V count. He was then referred to our out-patient department (9/9/43) and was advised to enter the sanatorium for a period of study. Admission to the sanatorium was on 9/21/43.

Physical examination revealed a well developed, fairly well nourished man whose only complaint was slight soreness in the right chest. Blood pressure was 130/82. Chest examination revealed hyperresonance to percussion and absent breath sounds in the upper half of the chest bilaterally. There were diminished breath sounds in the lower half of both sides. No rales were heard.

Laboratory Findings—All sputum examinations were negative for tubercle bacilli (two concentrated morning sputa, one 24 hour concentrate, three 48 hour concentrates, two 72 hour concentrates, and four cultures).

Roentgen-ray diagnosis was pulmonary cysts, bilateral (Fig. 1).

The impression of the staff was that we were dealing with a case of cystic disease of the lung. To further confirm this we carried out the following procedures:

1) On 9/27/43 a diagnostic artificial pneumothorax, right, was induced. Seven hundred cc. of air was introduced. Opening pressure readings were −7 −2 and the closing readings were −3 −1. X-rays were taken following this procedure (Fig. 2).

2) On the afternoon of 9/27/43 an iodized oil bronchogram on the right side was made.

3) On 10/4/43 600 cc. of air and 10 cc. of iodized oil were injected into the pleural space. The patient was placed in the horizontal position with head lower than the feet and then rolled forward and backward (being observed at the same time under the fluoroscope) to distribute the iodized oil over the surface of the pleura. X-rays were taken immediately (Figs. 3 and 4).

The final diagnosis was "cystic disease of the lung" and the patient was

discharged on 10/9/43 to be followed in the out-patient department. Because of the extensiveness of the cysts and the comparatively good clinical condition of the patient, it was thought that any surgical procedures were definitely contraindicated at this time.

SUMMARY

1) A resumé of the literature on cystic disease of the lung is presented. There is no general agreement as to whether this condition is congenital or acquired. Anatomically, there are two general types of cysts: Those of the first type are bronchial dilatations; the second group are cavities resembling emphysematous blebs lying subpleurally. The more common symptoms of cystic disease are: dyspnea, cyanosis, cough, cardiac palpitation and rarely hemoptysis. The main condition to be considered in differential diagnosis is spontaneous pneumothorax. Treatment in general is unsatisfactory.

2) The authors describe a new diagnostic procedure which may be employed in difficult cases. An artificial pneumothorax is established and 10 to 15 cc. of iodized oil injected into the pleural space. The patient is then manipulated around to allow the oil to spread out and come in contact with all the pleural surfaces. X-rays immediately following this are very distinctive.

3) A case is reported in which the above mentioned diagnostic procedure was used.

RESUMEN

1) Se presenta un resumen de la literatura relativa a la enfermedad quística del pulmón. No existe acuerdo general acerca de si este estado es congénito o adquirido. Hay dos tipos generales de quistes: los del primer tipo son dilataciones bronquiales; los del segundo consisten de cavernas subpleurales que se asemejan a ampollas enfisematosas. Los síntomas más comunes de enfermedad quística son: disnea, cianosis, tos, palpitación cardíaca y, raramente, hemoptisis. La condición más importante que debe considerarse en el diagnóstico diferencial es el neumotórax espontáneo. El tratamiento, en general, no es satisfactorio.

2) Los autores describen un nuevo procedimiento para el diagnóstico que puede emplearse en casos difíciles. Se lleva a cabo un neumotórax artificial y se inyecta de 10 a 15 cc. de aceite yodado en el espacio pleural. Se mueve al paciente de tal manera que el aceite se distribuya y alcance a estar en contacto con todas las superficies pleurales. Radiografías tomadas inmediatamente después de ésto resultan muy claras.

3) Se informa sobre un caso en el que se empleó el procedimiento mencionado para diagnosticar.

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Editor's Note: The original article's bibliography contained 107 references, which space did not permit printing.

The Paradox of Vocational Disability

LEOPOLD BRAHDY, M.D., F.C.C.P.
New York, New York

Insurance benefits or sick-pay during periods of vocational disability are becoming quite common. In tuberculosis it is not easy to decide at what time a patient should resume some gainful occupation and, later, at what time he is able to resume his regular work. After having made the decision, the physician is faced with the more difficult task of submitting a report, giving the reasons for his conclusions in language that the layman can understand. The physician cannot escape this duty. He should welcome it because these reports bring the opportunity to educate employers, insurance carriers and civic authorities on the role of rehabilitation in tuberculosis. It is only too true that "official agencies have consistently underestimated the significance of rehabilitation."[1] Perhaps this is in part because physicians have not explained its significance in specific individual cases. In order to get all agencies to take a sounder view, we must take advantage of opportunities in individual cases to reiterate the role of rehabilitation in prevention and treatment instead of merely proclaiming general principles and then expect laymen to apply them. Before teaching others, the physician himself must study the relationship of the disease to the patient and to his earning capacity. This is a complicated problem involving personal, social and economic factors but it is essentially a *medical* problem—a problem in functional diagnosis and therapy. The physician must think it through until he can express his reasons convincingly.

Recently, an ex-patient was referred to me because of disagreement between two physicians. One reported: This man has arrested tuberculosis; he is able to resume some work. The second physician agreed that the disease was arrested, but concluded that he was totally disabled from any gainful occupation. The reports of both physicians evidenced a desire to be accurate, but failed to take into account what "able to work" meant to that ex-patient. Their reasoning on the relationship of rehabilitation to disability was muddled. My own conclusion seemed to increase the confusion until my reasons were given consideration and then they were accepted. My conclusions were: "This man is able to do some light work or else to resume his usual work for short hours; however, he is totally disabled at present." This is the paradox of vocational disability. It is hoped that the exposition of the paradox in this

case will clarify the principles of rehabilitation in tuberculosis and will aid the physician in proper certification of disability in tuberculosis.

CASE REPORT

J. H. N., former hospital helper. Referred for decision as to whether he is totally disabled or partially disabled, or able to resume his occupation.

This man had six years of elementary schooling. After leaving school, he did various odd jobs until he was 18, when he was hired as a hospital helper. At the hospital he had been a steady and satisfactory worker for five years when it was discovered that he had active tuberculosis acquired in the course of his occupation. He was then 23 years old. He was admitted to a sanatorium. Complete bed rest was prescribed during the first months of his illness and later, when permitted to get out of bed, he was taught to do things slowly, to use a minimum of physical effort, to abstain from mental exertion and to avoid emotional reactions. As his condition improved, ordinary activity was permitted. Toward the end of his first year he worked in the sanatorium carpenter shop for an hour a day. He enjoyed doing this for the next four months, and then he was discharged from the sanatorium. After that he stayed at home, went out for a daily walk, but did no further work.

I saw him one year ago. X-rays and clinical investigation had shown that for some months his disease had been arrested. Under the mild activities of his daily routine he felt well. He desired to do some useful work, but was uncertain as to whether he was able or should even try to resume his regular duties as a hospital helper. "My kind of work was never easy and I know it is tougher now because of the shortage of help." He had applied for part-time work elsewhere but, he added: "They don't want me when I tell them I've had no job for four years on account of tuberculosis." At that time I told him either to get a part-time job or to undergo some training for a trade.

He began a course in free-hand drawing at a commercial art school. He has been going to this school for two hours a day, and hopes to qualify as an assistant to a comic strip artist, but he is vague as to this prospective occupation. He brought some of his school sketches; they showed some ability in copying, but no special talent.

As required by the Compensation Law for Occupational Diseases, he is receiving $14.25 a week from the hospital in which he had been employed as long as he is totally disabled. All necessary treatment must be provided. Those payments are discontinued when he has recovered his previous earning capacity.

A machine may stand idle for years and, if it has been kept clean and oiled, it can be set going again at a moment's notice. But a man on enforced rest for the same period undergoes profound physical, mental and emotional changes which, even though the disease is arrested, make him incapable of doing any useful work. These changes in a man are reversible; his capacity for physical exertion can be recovered; his psychic and emotional stability can be restored. The normal desire to earn his own living will then assert itself and, with proper encouragement, will become dominant. To

guide him toward this goal requires knowledge of how and when and what to do. This is vocational rehabilitation.

Vocational rehabilitation can be achieved only if it is under direct medical control and is supervised by persons acquainted with the technique of rehabilitation. Rehabilitation is treatment. It is a part of the treatment as necessary for a tuberculosis patient as prescribed rest, diet or pneumothorax. This man should be given vocational rehabilitation as part of the treatment of his disease. His treatment is incomplete until he has been rehabilitated.

Rehabilitation of tuberculosis patients is provided in many institutions and some, such as the Potts Institute and the Altro Work Shop, have this as their main purpose. In these specialized rehabilitation institutions results are excellent, but all patients are not suitable for admission to them. More such institutions are needed but it is no longer necessary or desirable that all patients receive rehabilitation by going there. Their methods have been formulated and published; they can now be applied by others. The staffs at these institutions have accomplished more; they have taught the medical profession that their institutional results can be improved by rehabilitating employees *within industry*. This means specialized rehabilitation institutions are not essential for those ex-patients who had definite occupations which they can resume in graded stages. For these ex-employees the best results are obtained when rehabilitation is done *in the same plant or office, at the same occupation in which they formerly worked*. In business and general industry that is not easy because the supervising personnel never had contact with sick or handicapped workers. The background of the supervisors may not be such that they can be taught rehabilitation principles very easily. In spite of this, the system of rehabilitation by assigning selected tasks within industry with shorter hours and longer rest periods is functioning well. In one field of employment conditions for rehabilitation are ideal—that field is in hospitals. The supervising personnel in a hospital is better informed on medical problems—they have a more sympathetic attitude toward their handicapped employees and they can quickly learn the methods of rehabilitation. In a hospital, medical supervision is more readily available. The variety of jobs in a hospital requires all grades of skill, intelligence and exertion. All this makes the task of rehabilitation, when undertaken by hospitals, less difficult than in any other industry.

In the case of J. H. N., cited above, his former employer is a large hospital with many jobs to be done. The hospital is now short of trained personnel. Here is a man with five years' experience in a job. Hospitals are now forced to train men in a few weeks for those jobs and put up with their lack of skill. Rehabilitation of an experi-

enced man is part of the national war effort. At present this man cannot tackle a full-time job. But with medical supervision of his assignments, he could now do much useful work and later could be guided back to full service. If he is not started on a light assignment, he can do no work at all. He will be useless to himself and to everyone else. He will continue to be an expense to the former employer, who must continue to pay his compensation. He and his family must continue to live on his meager compensation payments instead of on his normal earnings. We know that ex-patients who have been rehabilitated are less likely to have a recurrence than those who drift along in idleness. No new employer is likely to give him a supervised light job, except as a matter of philanthropy. As a matter of enlightened self-interest, the former employer who must pay his compensation should provide such a job.

CONCLUSION

This man, now 28 years old, has a partial disability, and is able to work short hours under medical control. He should be asisgned 3 hours' work every morning, six days a week for 3 months, after which his assignments probably can be steadily increased. This should be begun now, and until it is begun he is totally disabled.

SUMMARY

It is part of medical diagnosis when physicians decide a patient has recovered sufficiently to do light work or to do his regular work on part-time. Disability certification at this stage of tuberculosis is well formulated in the paradox "From a vocational viewpoint, totally disabled at the present time but capable of doing selected work." Such certification gives the physician the opportunity to explain the role of rehabilitation to employers, insurance carriers and civic authorities who require disability certificates in order to administer sick-pay and insurance benefits. Employers and administrators have not been sufficiently informed on this important factor for recovery. Physicians must use each case to reiterate that rehabilitation is part of treatment. If a job is made available in which the work, hours and the patient are under medical control, then the patient is able to do useful work and will probably progress rapidly toward completely restored working capacity. If such a job is not provided, he cannot do any work, continues totally disabled and progresses toward completely restored working capacity at a snail's pace. The author cites the case of a hospital worker to illustrate these principles.

Rehabilitation through medically controlled wage-earning work is most successful when done by the employer for whom the patient worked prior to his illness. The employers who have the best

facilities and opportunities for rehabilitating their former employees are our hospitals. Special institutions for rehabilitation are valuable especially because they have taught us that the major part of rehabilitation must be accomplished within industry.

RESUMEN

Es parte del diagnóstico el que los médicos decidan si un paciente se ha restablecido lo suficiente para hacer un trabajo liviano o para reasumir parcialmente su ocupación ordinaria. La certificación de incapacidad en este período de la tuberculosis está bien formulada en la paradoja: "Desde el punto de vista vocacional totalmente incapacitado al presente, pero capacitado para hacer un trabajo seleccionado." Tal certificación ofrece al médico la oportunidad de explicar el papel que desempeña la rehabilitación a los patrones, agentes de Seguros y autoridades cívicas que requieran certificados de incapacidad para poder pagar los salarios de enfermos o los beneficios del Seguro. Los patrones y los administradores no han sido suficientemente informados sobre este factor importante para el restablecimiento. Los médicos deben aprovechar cada caso para insistir que la rehabilitación es parte del tratamiento. Si se presenta un empleo en el que la clase de trabajo, las horas de labor y el paciente quedan bajo dirección médica, entonces el paciente está capacitado para prestar un servicio útil, y probablemente progresará con rapidez hacia la completa restauración de su capacidad de trabajo. Si no se proporciona tal empleo, el paciente no puede trabajar en lo absoluto, continúa totalmente incapacitado y avanza a paso de tortuga hacia la completa restauración de su capacidad para trabajar. Cita el autor el caso de un empleado de hospital para ilustrar estos principios.

La rehabilitación mediante el trabajo remunerado bajo superintendencia médica obtiene mayor éxito cuando la lleva a cabo el patrón para quien trabajaba el paciente antes de su enfermedad. Los patrones que tienen mejores facilidades y oportunidades para rehabilitar a sus ex-empleados son nuestros hospitales. Las instituciones especiales de rehabilitación son valiosas especialmente porque ellas nos han enseñado que la mayor parte de la rehabilitación debe llevarse a cabo dentro de la industria.

REFERENCE

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EDITORIAL

THE TUBERCULOSIS PROBLEM IN PUERTO RICO

In Puerto Rico, tuberculosis stands unrivalled as Public Enemy Number One. It causes nearly one-half of all deaths of persons between nineteen and thirty-five years of age. Because it is responsible for widespread disability and loss of life among young parents and breadwinners, it constitutes an economic as well as a public health problem.

After twenty years of steadily increasing tuberculosis mortality, the rate in 1933 reached 337, one of the highest in the civilized world; and it seemed that the disease was becoming epidemic. The alarmed Public Health authorities launched an intensive campaign against it, and in 1934 tripled the number of beds for open cases, bringing the total to 1,500. This number was still extremely inadequate, since our average of more than 5,000 tuberculosis deaths annually called for more than 10,000 beds, according to the standards of the National Tuberculosis Association.

The urgent need was then, and is now, isolation of open cases. In the homes isolation is impossible. Puerto Rico is an agricultural country, densely populated and poverty stricken. Most of the inhabitants live in slums where huts are crowded together, families are crowded within the huts, sanitary facilities are meager in the extreme, and everything favors the spread of contagious diseases. Without hospital beds it is foolish to preach isolation, when, in the home, five or six persons, including the patient, must of necessity live in one small room.

In the hope of controlling spread of tuberculosis from the open cases who could not be isolated, the next step undertaken was the application of artificial pneumothorax to ambulant patients. Simultaneously, the early diagnosis campaign was greatly intensified, especially through the examination of contacts of open cases. The first pneumothorax dispensaries were opened in 1935. By the end of the fourth year nearly 2,000 patients were receiving pneumothorax while living at home. Also, 147,000 persons had been examined for tuberculosis in Health Department Clinics.

For the first time since reliable statistics were available, the mortality curve started on a sustained downward course, and in six years after the beginning of the campaign, the mortality rate dropped twenty per cent.

It is disheartening to report, however, that after such an effort our tuberculosis death rate in Puerto Rico is still 245 (in 1941), five and one-half times the rate in continental United States. Pneumothorax can control a certain number of open cases, but the majority remain a public health menace unless hospitalized.

Were we to provide the required number of beds, the cost for the first five years would run into some fifty millions of dollars—an impossible undertaking, considering our limited means and other pressing needs of the island. Our only hope is to obtain Federal aid. The Puerto Rico Chapter of the American College of Chest Physicians has made an appeal for such help to Senator Dennis Chavez, president of a Congressional Committee recently sent to the island to investigate social and economic conditions. We hope that this plea will be effective.

Even in the midst of a desperate struggle for the survival of democracy, we cannot help but think that the tuberculosis problem of Puerto Rico could be solved with less cost than building one battleship. Perhaps it is a sinful thought at this moment, since the objectives of the war are so much more desirable than life; but it is a good thought to keep in mind until after the war, when human needs will have a new perspective and human values will rise to higher levels.

—J. Rodriguez Pastor, M.D., F.C.C.P.

Report of the
Council on Pan American Affairs

The Council on Pan American Affairs of the College is very happy to report that the Argentine Chapter was founded April 29, this being the fifth Latin American chapter to be founded. The officers of the chapter are: President, Dr. Gumersindo Sayago; vice-president, Dr. Raul Vaccarezza; secretary-treasurer, Dr. Juan Carlos Rey.

The next chapter will unquestionably be the Peruvian because our governor for Peru, Dr. Ovidio Garcia-Rosell, came to Chicago bringing with him sixteen applications for membership which are sufficient for the formation of a chapter.

Reviewing the organization of Latin American chapters, the first was founded in Cuba in December, 1940, the second in Brazil in November, 1942, the third in Puerto Rico in January, 1943, the fourth in Mexico in September, 1943, and now the fifth in Buenos Aires in April, 1944. Thus the College now has chapters in four Latin American countries, and a chapter in Puerto Rico; and a chapter will very shortly be formed in Peru. Governors are at work on the organization of chapters in Chile, Colombia, Ecuador, Panama and Venezuela. Contacts are being established with chest physicians of the remaining countries, and invitations to join the College extended to outstanding specialists.

It is most gratifying that we have had such good attendance by Latin American colleagues at the present meeting, despite the difficulties of transportation. The president of the Cuban Chapter, Dr. Teodosio Valledor, had definitely planned to come to the meeting to represent the Cuban Chapter but was unable to complete his arrangements for transportation. The Brazilian Chapter had expected to send a delegate but was likewise unable to secure the necessary priority at the last minute. The Puerto Rico Chapter sent an official delegate, Dr. Velasquez. The Mexican Chapter has been exceptionally well represented by its president, Dr. Donato Alarcon; its vice-president, Dr. Cosio Villegas; its secretary, Dr. Octavio Bandala, and several other members. Chile is represented by Dr. Enrique Garcia Suarez and Dr. Julio Urrutia, who bring letters from our governor for Chile, Dr. Orrego Puelma.

The Council on Pan American Affairs held a successful meeting last night at which not only a number of our Latin American colleagues but both the incoming and outgoing presidents of the College, the secretary and the executive secretary, the editor-in-chief of *Diseases of the Chest*, the chairman of the Membership Committee, General Marietta, and one of our vice-presidents, Dr. Overholt, were present. Furthermore, Dr. William E. Ogden of Toronto, governor of the College for Canada, was also in attendance and reported that Canadian membership of the College has grown in the past year or so from 8 to between 30 and 40.

One other important matter remains to be mentioned in the Report of the Council on Pan American Affairs, and that is the survey of opportunities in the various sanatoria and hospitals for chest diseases in the United States for residents and postgraduate students from the Latin American countries. This survey was conducted in a very efficient manner by Mr. Kornfeld. A total of 152 institutions returned questionnaires and 92 stated that they would be willing to accept one or more physicians

from the other American republics for postgraduate medical education in tuberculosis. The following information has been compiled from the 92 informative questionnaires: Sixty-nine (69) institutions indicated that they had facilities for housing these physicians. All of the sanatoria indicated that they had facilities for teaching diagnosis and treatment of diseases of the chest, and in many instances, they have indicated the various services which are available at the institutions or in connection with some medical school. In 74 institutions, an out-patient clinic is affiliated with the sanatorium or this service is made available in a nearby city. Physicians would be eligible to receive a stipend from 35 of the institutions in varying amounts ranging up to $200.00 per month. In 30 of the institutions suitable housing facilities can be found for physicians who desire to live outside of the sanatorium. The cost for rental and maintenance varies greatly according to the location of the institutions. In 18 of the institutions the Spanish language is spoken; 1 institution reports that Portuguese is spoken, and in 26 institutions the French language is spoken. In 75 of the institutions it was reported that the physicians would be able to obtain training in case-finding and other public health activities pertaining to tuberculosis and diseases of the chest. The minimum number of beds in the 92 institutions with which this report is concerned is 70, and the maximum number of beds is 1219. The number of physicians on the staff as well as the number of nurses is reported on each of the questionnaires. In some cases individual letters have accompanied the questionnaires, and copies of these letters are included in this report.

Several men have already been placed with the aid of the information obtained in this survey, and we stand ready to help the Pan American Sanitary Bureau, the Office of the Coordinator of Inter-American Affairs and the State Department, as well as private individuals in the placement of postgraduate students and physicians interested in diseases of the chest.

Chevalier L. Jackson, M.D., F.C.C.P.,
Chairman,
Philadelphia, Pennsylvania

Report of the Board of Examiners

The first examination for Fellowship in the College was conducted at Atlantic City, New Jersey, in June, 1942. Twenty-five candidates for Fellowship took the oral examination; 24 passed, 1 failed.

The first written examination was conducted by the Board of Examiners of the College at various cities throughout the United States in January, 1943. Twenty candidates took this written examination of which 16 passed, 4 failed.

The next written examination was given in Cincinnati, Ohio, and at other cities throughout the United States in November, 1943. Ten candidates took this written examination for Fellowship, all of whom successfully passed.

In June, 1944, 32 candidates took the written examination at Chicago and at other cities throughout the United States. The results of this last examination have not as yet been tabulated.

	Candidates	*Passed*	*Failed*
June, 1942	25	24	1
January, 1943	20	16	4
November, 1943	10	10	0
June, 1944	32*	..	..
	87	50	5

*Results of the June, 1944, examinations have not as yet been tabulated.

George G. Ornstein, M.D., F.C.C.P.,
Chairman,
New York, New York

COLLEGE NEWS

PRELIMINARY SCIENTIFIC PROGRAM SPONSORED BY THE COLORADO MEMBERS, AMERICAN COLLEGE OF CHEST PHYSICIANS

Cosmopolitan Hotel—Denver, Colorado
Wednesday, Sept. 27, 1944

9:00 a. m.—Scientific Session.

"Pulmonary Complications of Thrombophlebitis," Douglas Deeds, M.D., Denver, Colorado. Discussant: Major Robert Woodruff, M.C., U.S.A., Denver, Colorado.

"Chemotherapy in Experimental Tuberculosis (A Review)," H. Corwin Hinshaw, M.D., and William H. Feldman, D.V.M., Mayo Clinic, Rochester, Minnesota. Discussant: Arthur Rest, M.D., F.C.C.P., Spivak, Colorado.

"Cystic Disease of the Lungs," Major Robert Liggett, M.C., U.S.A., Denver, Colorado.

"Pulmonary Resection in the Treatment of Tuberculosis," Capt. Charles V. Demong, M.C., U.S.A., Denver, Colorado.

12:00 Noon—Luncheon.

"The Role of the Chest Specialist in the Control of Tuberculosis," Louis Mark, M.D., F.A.C.P., F.C.C.P., Columbus, Ohio.

2:00 p. m.—Scientific Session.

"The Synergistic Relationship Between Mycotic and Tuberculous Infections of the Lung," Alvis E. Greer, M.D., F.A.C.P., F.C.C.P., Houston, Texas.

"Minimal Tuberculosis," Capt. W. H. Roper, M.C., U.S.A., Denver, Colo.

"Lung Resection for Chronic Pulmonary Infection," Richard Davison, M.D., F.A.C.S., F.C.C.P., Chicago, Illinois.

"Diasone in the Treatment of Pulmonary Tuberculosis," Charles Kaufman, M.D., F.C.C.P., Denver, Colorado.

General Arrangements Committee

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SCIENTIFIC PROGRAM SPONSORED BY WISCONSIN MEMBERS AMERICAN COLLEGE OF CHEST PHYSICIANS

Schroeder Hotel—Milwaukee, Wisconsin
Sunday, Sept. 17, 1944

2:00 p. m.—Scientific Session, Pere Marquette Room.

John K. Shumate, M.D., F.C.C.P., Madison, Wisconsin, presiding.

"Pregnancy in Tuberculosis," Fred M. F. Meixner, M.D., F.A.C.S., F.C.C.P., Peoria, Illinois. Discussants: Arthur S. Webb, M.D., F.C.C.P., Glen Ellyn, Illinois; R. W. Roethke, M.D., Milwaukee, Wisconsin.

"Tuberculosis Control in General Hospitals," Minas Joannides, M.D., F.A.C.S., F.C.C.P., Chicago Illinois. Discussion to be opened by Otto C. Schlack, M.D., F.C.C.P., Oak Forest, Illinois.

"Tuberculosis of the Nasopharynx," A. R. Hollender, M.D., F.A.C.S., Chicago, Illinois. Discussants: William E. Grove, M.D., Milwaukee, Wisconsin; Edwin R. Levine, M.D., F.C.C.P., Chicago, Illinois.

"Surgical Management of Empyema," Richard Davison, M.D., F.A.C.S., F.C.C.P., Chicago, Illinois. Discussants: Otto L. Bettag, M.D., F.C.C.P., Pontiac, Illinois; John D. Steele, Jr., M.D., Milwaukee, Wisconsin; Karl Schlaepfer, M.D., Milwaukee, Wisconsin.

6:00 p. m.—Dinner Meeting, Parlor A.

Carl O. Schaefer, M.D., F.A.C.S., F.C.C.P., Racine, Wisconsin, presiding.

"The Medical Profession and the Control of Tuberculosis," Jay Arthur Myers, M.D., F.A.C.P., F.C.C.P., Minneapolis, Minnesota, President, American College of Chest Physicians.

Organization of Wisconsin Chapter, American College of Chest Physicians.

8:00 p. m.—X-Ray Conference, Pere Marquette Room.

H. H. Christensen, M.D., F.A.C.S., F.C.C.P., Wausau, Wisconsin, presiding.

X-ray films of interest will be shown at this conference.

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Leonard W. Moody, M.D., F.C.C.P., Bayfield
Bert L. Jones, M.D., F.C.C.P., Wood

PENNSYLVANIA CHAPTER

The Annual Meeting of the Pennsylvania Chapter of the American College of Chest Physicians will be held Tuesday evening, September 19, 1944, at Pittsburgh, Pennsylvania. This meeting is being held in conjunction with the annual meeting of the Pennsylvania Medical Association which meets at Pittsburgh, September 19-21.

Plans have been made for a short business meeting, and an x-ray conference to be followed by the annual dinner of the chapter. For further particulars concerning this meeting, please communicate with Dr. Edward Lebovitz, F.C.C.P., Secretary-Treasurer, Pennsylvania Chapter, American College of Chest Physicians, 617 Jenkins Building, Pittsburgh, Pennsylvania.

MICHIGAN CHAPTER

The Michigan Chapter of the American College of Chest Physicians will hold its annual meeting at the Pantlind Hotel, Grand Rapids, Michigan, on September 28, 1944, in conjunction with the annual meeting of the Michigan State Medical Society, which meets at Grand Rapids, September 27-29.

The following program has been arranged:

"Symposium on Virus Pneumonia," Norman E. Clarke, M.D., F.C.C.P., and Oliver Marcotte, M.D., F.C.C.P., Detroit.

"The Physician's Role in Tuberculosis," Jay Arthur Myers, M.D., F.C.C.P., Minneapolis, Minnesota, President, American College of Chest Physicians.

For further particulars concerning this meeting, please communicate with Dr. William P. Chester, F.C.C.P., Secretary-Treasurer, Michigan Chapter, American College of Chest Physicians, 2916 Seminole Avenue, Detroit, Michigan.

INDIANA CHAPTER

The Indiana Chapter, American College of Chest Physicians, will hold a luncheon meeting at the Murat Temple, Indianapolis, on Tuesday, Oct. 3, 1944, in connection with the annual meeting of the Indiana State Medical Association.

Dr. Paul H. Holinger, F.C.C.P., Secretary-Treasurer of the College, will be the guest speaker and will present a paper on "Bronchoscopic Diagnosis of Bronchial Tumors." This will be followed by a technicolor movie on "Lesions of the Bronchial Tract," which is being sponsored by the Anti-Tuberculosis Committee of the Indiana State Medical Association.

An X-Ray Conference and election of officers will follow.

For further particulars concerning this meeting, please communicate with Dr. Hubert B. Pirkle, F.C.C.P., Secretary-Treasurer, Indiana Chapter, Superintendent, Indiana State Sanatorium, Rockville, Indiana.

PROGRAM SOUTHERN CHAPTER AMERICAN COLLEGE OF CHEST PHYSICIANS

November 13-14, 1944

Meeting Conjointly With the Southern Medical Association
November 13-16, 1944

The annual meeting of the Southern Chapter, American College of Chest Physicians, meeting conjointly with the Southern Medical Association, will be held at St. Louis, Missouri, November 13-14.

Program

Monday, November 13

9:30 A. M. ..... DeSoto Hotel

Registration.

10:00 A. M. ..... DeSoto Hotel

Semi-Annual Meeting, Board of Regents, J. C. Placak, M.D., F.C.C.P., Cleveland, Ohio, Chairman, Board of Regents, presiding.

12:30 P. M. ..... DeSoto Hotel

Luncheon, Board of Regents and Board of Governors (Fellows and guests invited), Jay Arthur Myers, M.D., F.C.C.P., Minneapolis, Minnesota, President, American College of Chest Physicians, presiding.

2:00 P. M. ..... St. Louis Municipal Auditorium

Scientific Session, Paul H. Ringer, M.D., F.C.C.P., Asheville, North Carolina, President, Southern Chapter, presiding.

"The Unexpandable Lung," Sydney Jacobs, M.D., F.C.C.P., New Orleans, Louisiana. Discussion to be opened by Carl C. Aven, M.D., F.C.C.P., Atlanta, Georgia.

"Primary Carcinoma of the Lung—Ten-Year Follow-Up," William F. Rienhoff, Jr., M.D., Baltimore, Maryland. Discussion to be opened by Evarts Graham, M.D., F.C.C.P., St. Louis, Missouri.

"Rehabilitation of Cases of Lung Resection," Lt. Col. Brian B. Blades, M.C., Washington, D. C. Discussants: *Alton Ochsner, M.D., F.C.C.P., New Orleans, Louisiana, and Maurice G. Buckles, M.D., F.C.C.P., Louisville, Kentucky.

"What Shall We Do With Silent and Masquerading Chest Lesions?", Richard H. Overholt, M.D., F.C.C.P., Brookline, Massachusetts. Discussion to be opened by Duane Carr, M.D., F.C.C.P., Memphis, Tennessee.

6:30 P. M. ..... DeSoto Hotel

Cocktail Party sponsored by the Missouri Chapter, American College of Chest Physicians.

7:15 P. M. ..... DeSoto Hotel

President's Dinner (informal), *Walter Vest, M.D., F.C.C.P., Huntington, West Virginia, Toastmaster. Introduction of Jay Arthur Myers, M.D., F.C.C.P., Minneapolis, Minnesota, President, American College of Chest Physicians, and Charles M. Hendricks, M.D., F.C.C.P., El Paso, Texas, President-Elect, American College of Chest Physicians.

Guest Speaker: Herman E. Hilleboe, M.D., F.C.C.P., Washington, D. C., Medical Director, United States Public Health Service.

President's Address: Paul H. Ringer, M.D., F.C.C.P., Asheville, North Carolina, President, Southern Chapter, American College of Chest Physicians.

Tuesday, November 14

9:00 A. M. ..... St. Louis Municipal Auditorium

Scientific Session, Herbert L. Mantz, M.D., F.C.C.P., Kansas City, Missouri, presiding.

"Atypical Pneumonia Resembling Pulmonary Tuberculosis," Major Walter L. Nalls, M.C., F.C.C.P., Washington, D. C. Discussion to be opened by Dean B. Cole, M.D., F.C.C.P., Richmond, Virginia.

"Hedblom's Syndrome: Acute Primary Diaphragmitis," Minas Joannides, M.D., F.C.C.P., Chicago, Illinois.

"Tuberculous Empyema," George G. Ornstein, M.D., F.C.C.P., New York, New York. Discussants: H. I. Spector, M.D., F.C.C.P., St. Louis, Missouri, and Karl Schaffle, M.D., F.C.C.P., Asheville, North Carolina.

"Pneumoconiosis, Especially as Found in the Metal and Granite Trades," O. A. Sander, M.D., Milwaukee, Wisconsin.

12:30 P. M. ..... DeSoto Hotel

Luncheon to be followed by Business Meeting, Paul A. Ringer, M.D.,

F.C.C.P., Asheville, North Carolina, President, Southern Chapter, American College of Chest Physicians, presiding.

2:00 P. M. ..... DeSoto Hotel

X-Ray Conference, H. Frank Carman, M.D., F.C.C.P., Dallas, Texas, presiding.

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*Invited, acceptance not received at time of publication.

Officers and Committees, Southern Chapter

President, Paul H. Ringer, M.D., F.C.C.P., Asheville, North Carolina
First Vice-President, Alvis E. Greer, M.D., F.C.C.P., Houston, Texas
Second Vice-President, Carl C. Aven, M.D., F.C.C.P., Atlanta, Georgia
Secretary-Treasurer, Benjamin L. Brock, M.D., F.C.C.P., Waverly Hills, Kentucky

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Merle D. Bonner, M.D., F.C.C.P., Jamestown, North Carolina

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Several of the hotels are already booked to capacity and others are filling up rapidly. For reservations, address Hotel Committee, Southern Medical Association, 901 Syndicate Trust Building, St. Louis 1, Missouri.

For further particulars concerning this meeting, please communicate with Benjamin L. Brock, M.D., F.C.C.P., Secretary-Treasurer, Southern Chapter, Waverly Hills Sanatorium, Waverly Hills, Kentucky, or H. I. Spector, M.D., F.C.C.P., Chairman, Arrangements Committee, 622 University Club Building, St. Louis 3, Missouri.

NEW JERSEY CHAPTER

The officials of the New Jersey Chapter, American College of Chest Physicians, met at the Essex House, Newark, New Jersey, on June 28, 1944, and at the Shonghum Mountain Sanatorium, Morris Plains, New Jersey, on July 26, 1944. Plans for the Fall Meeting of the chapter were discussed, and it was decided to hold the meeting at the Valley View Sanatorium, Paterson, New Jersey, on November 16, 1944, at 8:30 p. m. The members of the Passaic County Medical Society will be the guests of the chapter. An interesting program on diseases of the chest is being arranged.

For further particulars concerning this meeting, please communicate with Dr. Harold S. Hatch, F.C.C.P., Secretary-Treasurer, New Jersey Chapter, Shonghum Mountain Sanatorium, Morristown, New Jersey.

CALIFORNIA CHAPTER

Dr. Seymour M. Farber, F.C.C.P., San Francisco, has been appointed secretary-treasurer of the California Chapter, American College of Chest Physicians, succeeding Dr. J. J. Singer, F.C.C.P., who resigned because of ill health. Dr. Stephen A. Parowski, F.C.C.P., San Diego, is President of the chapter, and Dr. Charles L. Ianne, F.C.C.P., Stanford University, is Vice-President.

PRESIDENT-ELECT OF CUBA RECEIVES COLLEGE OFFICIALS

A delegation of the Cuban Chapter of the American College of Chest Physicians was received by the President-Elect of Cuba, Dr. Ramon Grau San Martin. This delegation was comprised of Dr. Octavio Rivero, President, Dr. Rene Garcia Mendoza, Vice-President, Dr. Orfilio Suarez de Bustamante, Secretary-Treasurer, and Dr. Antonio Navarrete, Regent. Introductions were made by Dr. Rivero, a close friend of Dr. Grau, President-Elect of the Republic of Cuba.

A congratulatory message from Dr. Jay Arthur Myers, President of the American College of Chest Physicians, was delivered to Dr. Grau on behalf of the members of the College. Dr. Grau expressed his thanks and extends to the members of the College his appreciation, goodwill, and sympathy for the College and its work.

CUBAN CHAPTER ELECTS OFFICERS

The Cuban Chapter of the College held its annual election of officers on July 6 and the following physicians were elected to office:

President, Dr. Octavio Rivero, F.C.C.P., Havana
Vice-President, Dr. Rene Garcia Mendoza, Havana
Secretary-Treasurer, Dr. Orfilio Suarez de Bustamante, Havana

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COLLEGE NEWS NOTES

Dr. Teodosio Valledor, Havana, and Dr. Rene Garcia Mendoza, Havana, have been appointed Medical Director and Assistant Medical Director, respectively, of the new Children's Tuberculosis Sanatorium recently completed in Havana. Both of these physicians won their appointments through competitive exercises.

Dr. Chevalier L. Jackson, F.C.C.P., Chairman of the Council on Pan American Affairs, has been appointed official delegate of the American Academy of Ophthalmology and Otolaryngology to be held at Montevideo, Uruguay, in October.

Reprints have been received from Dr. Jose Silveira, Bahia, Brazil, and added to the College library.

Dr. Shu-Fan Li, F.C.C.P., Governor of the College for China, spoke at Plummer Hall, Mayo Clinic, Rochester, Minnesota, on June 19. Dr. Li discussed "The Medical Problems of South China." Dr. Li was in Hong Kong at the time of the Japanese invasion of Hong Kong and is at present visiting in this country. Dr. Li was the first Minister of Health of China and he is Director and Chief Surgeon of the Hong Kong Hospital.

Dr. George G. Ornstein, F.C.C.P., was guest speaker at the First National Congress of Tuberculosis and Silicosis held at Mexico City, July 23-29, 1944. Dr. Ornstein presented a paper on "Pathological Aspects of Tuberculosis."

Dr. J. Winthrop Peabody, F.C.C.P., Regent of the American College of Chest Physicians, will present a paper on "Post-war Planning for Tuberculosis Control in General Hospitals" before the annual meeting of the American Hospital Association, October 1, 1944, at Cleveland, Ohio. Dr. Joseph C. Placak, F.C.C.P., Chairman of the Board of Regents of the College, will discuss this paper.

The following members of the American College of Chest Physicians have been elected as Section Officers of the American Medical Association for 1944-45: Alton Ochsner, M.D., F.C.C.P., New Orleans, Louisiana, Secretary, Section on General Surgery. Louis H. Clerf, M.D., F.C.C.P., Philadelphia, Pennsylvania, Henry B. Orton, M.D., Newark, New Jersey, and Fletcher D. Woodward, M.D., Charlottesville, Virginia, have been elected Chairman, Vice-Chairman and Secretary, respectively, of the Section on Laryngology, Otology and Rhinology.

The following members of the College in Texas will hold an office or serve as a member of a committee of the Texas State Medical Society for the ensuing year: *Chairman, Board of Trustees:* Sam E. Thompson, M.D., F.C.C.P., Kerrville, President, Texas Chapter, American College of Chest Physicians; *Member, Board of Trustees:* J. B. McKnight, M.D., F.C.C.P., Sanatorium, Governor of the College for the State of Texas; *Chairman, Committee on Postgraduate Medical Education:* Felix P. Miller, M.D., F.C.C.P., El Paso; *Chairman, Tuberculosis Committee:* R. G. McCorkle, M.D., F.C.C.P., San Antonio; *Member, Tuberculosis Committee:* R. B. Homan, Jr., M.D., F.C.C.P., El Paso; *Chairman, Section on Public Health:* Victor E. Schulze, M.D., San Angelo; *Member, Committee on Library Endowment:* Orville E. Egbert, M.D., F.C.C.P., El Paso; *Members, Committee on Public Relations:* Felix P. Miller, M.D., F.C.C.P., El Paso, and Victor E. Schulze, M.D., San Angelo.

Dr. John C. Sharp, F.C.C.P., Salinas, California, Governor of the American College of Chest Physicians for the State of California, was elected Vice-President of the Association of Western Hospitals at their annual meeting held at San Francisco, July 5.

Dr. Evarts R. Graham, F.C.C.P., St. Louis, Missouri, was appointed a member of the Committee to Study and Survey American Hospitals and their Post-war Expansion Needs. The study and survey will be financed by a grant of $35,000 each by the Commonwealth Fund of New York, the National Foundation for Infantile Paralysis, and the W. K. Kellogg Foundation. The American Hospital Association will make an additional contribution of $15,000 if it becomes necessary. It is hoped that the study will determine the adequacy of distribution of present hospital facilities and the best method for insuring hospital care and such facilities to all citizens.

Dr. Walter E. Vest, F.C.C.P., Huntington, West Virginia, was elected Vice-President of the American Geriatrics Society at their annual meeting held at New York City, June 8-10. Dr. Vest is a member of the Council on Military Affairs and Public Health of the American College of Chest Physicians.

Dr. Martin H. Collier, F.C.C.P., Grenloch, New Jersey, was elected President of the State Board of Health of New Jersey. Dr. Collier is Chairman of the Committee on General Management and Rehabilitation of Diseases of the Chest of the American College of Chest Physicians and he served as the first President of the New Jersey Chapter of the College, 1940-41.

Dr. Charles P. Bailey, F.C.C.P., Jenkintown, Pennsylvania, will present a paper before the General Assembly of the Pennsylvania State Medical Society at their annual meeting to be held at Hotel William Penn, Pittsburgh, Wednesday morning, September 20, at 9:30 a. m. Dr. Bailey's subject will be "Lobectomy and Pneumonectomy in Modern Medicine."

Dr. Nelson Mercer, F.C.C.P., has been appointed Chief Medical Officer, Tuberculosis Division, Gallinger Municipal Hospital, Washington, D. C. He succeeds Charles P. Cake, M.D., F.C.C.P., who resigned this position to accept a position with the U. S. Public Health Service. Dr. Cake is stationed at the Marine Hospital, Staten Island, New York.

Dr. Fred M. F. Meixner, F.C.C.P., Peoria, Illinois, President of the Illinois Chapter, American College of Chest Physicians, has been appointed Chairman of the Committee on Tuberculosis of the Illinois State Medical Society.

Dr. David B. Gregg has been appointed clinic coordinator and assistant physician at Pinehaven Sanatorium, Charleston, South Carolina. Dr. Gregg was formerly resident at the State Tuberculosis Sanatorium, State Park, South Carolina.

Dr. David McCullough, F.C.C.P., has been appointed superintendent of the Kerrville State Sanatorium, Kerrville, Texas.

Dr. John Srail, F.C.C.P., has been appointed superintendent and medical director of the Oakhurst Sanatorium, Elma, Washington.

Dr. Alexander S. Mack has been appointed superintendent of the William Roche Memorial Hospital, Toledo, Ohio. Dr. Paul M. Holmes, F.C.C.P., is President of the Board of Trustees.

Dr. Frank B. Stafford, F.C.C.P., has been appointed superintendent of the Blue Ridge Sanatorium, Charlottesville, Virginia. Dr. Stafford had served as assistant superintendent of the sanatorium since its opening in 1920.

Dr. Charles K. Petter, F.C.C.P., has been elected President of the Lake County Tuberculosis Society, Lake County, Illinois.

A symposium on cancer of the lung was presented by Dr. Chevalier L. Jackson, F.C.C.P., and Dr. W. Emory Burnett before the Dauphin County Medical Society at the Harrisburg Academy of Medicine, Harrisburg, Pennsylvania, on May 16.

Lt. Comdr. Thomas E. Newell, F.C.C.P., Dayton, recently spent a fifteen-day leave at home after almost two years in the South Pacific with the First Marine Division.

Dr. H. B. Morgan, F.C.C.P., Ware Shoals, South Carolina, was the guest speaker at the meeting of the Abbeville County Medical Society on June 15. Dr. Morgan spoke on "Non-Tuberculous Diseases of the Chest."

Dr. John B. Floyd, F.C.C.P., Louisville, Kentucky, resigned his position as Director of Tuberculosis Control of the Kentucky State Department of Health.

The memory of the late Dr. R. B. Homan, Sr., El Paso, Texas, was honored on July 11 by the dedication of the Homan Solarium at the El Paso City-County Hospital. The solarium was built with funds supplied by friends of Dr. Homan when, at the time of his death, his family requested that money ordinarily spent for flowers be donated instead to the Tuberculosis Society for the perpetuation of Dr. Homan's work.

Major Frank P. Coleman has been appointed chief of surgical service at the McGuire General Hospital, Richmond, Virginia.

The American College of Surgeons has cancelled its annual Clinical Congress which was scheduled to have been held at Chicago, October 24-27, 1944.

COLLEGE OFFICERS

OFFICERS

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Minneapolis, Minnesota

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No. 3	C. Howard Marcy, M.D., Pittsburgh, Pennsylvania	1945
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No. 16	Gumersindo Sayago, M.D., Cordoba, Argentina	1946
No. 17	Donato G. Alarcon, M.D., Mexico City, Mexico	1946
No. 18	Affonso MacDowell, M.D., Rio de Janeiro, Brazil	1947
No. 19	J. Rodriguez Pastor, M.D., Santurce, Puerto Rico	1947
No. 20	William E. Ogden, Toronto, Ontario, Canada	1947
	Murray Kornfeld, Chicago, Illinois, Executive Secretary	

BOARD OF GOVERNORS

State	*Governor*	*City*	*Term Expires*
Alabama	L. Orton Davenport, M.D.	Birmingham	1945
Arizona	Howell Randolph, M.D.	Phoenix	1947
Arkansas	Jesse Dean Riley, M.D.	State Sanatorium	1947
California	John C. Sharp, M.D.	Salinas	1945
Colorado	Arnold Minnig, M.D.	Denver	1947
Connecticut	Lyman R. Morse, M.D.	Hartford	1945
Delaware	Lawrence D. Phillips, M.D.	Marshalltown	1946
District of Columbia	Wm. D. Tewksbury, M.D.	Washington	1947
Florida	M. Jay Flipse, M.D.	Miami	1947
Georgia	James A. Redfearn, M.D.	Albany	1945
Idaho	Orval F. Swindell, M.D.	Boise	1947
Illinois	Robert K. Campbell, M.D.	Springfield	1945
Indiana	Jerome V. Pace, M.D.	New Albany	1946
Iowa	J. Carl Painter, M.D.	Dubuque	1946
Kansas	Arthur L. Ashmore, M.D.	Wichita	1947

BOARD OF GOVERNORS *(Cont.)*

State	*Governor*	*City*	*Term Expires*
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Louisiana	Louis A. Monte, M.D.	New Orleans	1945
Maine	Francis J. Welch, M.D.	Portland	1947
Maryland	Victor F. Cullen, M.D.	State Sanatorium	1947
Massachusetts	Hubert A. Boyle, M.D.	Middleton	1945
Michigan	*William A. Hudson, M.D.	Detroit	1947
Minnesota	G. Arvid Hedberg, M.D.	Nopeming	1945
Mississippi	John S. Harter, M.D.	Sanatorium	1945
Missouri	Herbert L. Mantz, M.D.	Kansas City	1945
Montana	Frank I. Terrill, M.D.	Deer Lodge	1946
Nebraska	John F. Allen, M.D.	Omaha	1946
New Hampshire	Robert B. Kerr, M.D.	Manchester	1946
New Jersey	Marcus W. Newcomb, M.D.	Browns Mills	1945
New Mexico	William H. Thearle, M.D.	Albuquerque	1946
New York	George Foster Herben, M.D.	Yonkers	1945
North Carolina	Merle D. Bonner, M.D.	Jamestown	1947
Ohio	John H. Skavlem, M.D.	Cincinnati	1947
Oklahoma	Robert M. Shepard, M.D.	Tulsa	1946
Oregon	James M. Odell, M.D.	The Dalles	1946
Pennsylvania	Victor M. Leffingwell, M.D.	Sharon	1946
Rhode Island	U. E. Zambarano, M.D.	Wallum Lake	1946
South Carolina	Richard K. Brown, M.D.	Greenville	1946
Tennessee	Raymond R. Crowe, M.D.	Nashville	1945
Texas	J. B. McKnight, M.D.	Sanatorium	1947
Utah	Wm. C. Walker, M.D.	Salt Lake City	1945
Vermont	Louis Benson, M.D.	Pittsford	1945
Virginia	Dean B. Cole, M.D.	Richmond	1947
Washington	John E. Nelson, M.D.	Seattle	1947
West Virginia	George R. Maxwell, M.D.	Morgantown	1947
Wisconsin	Carl O. Schaefer, M.D.	Racine	1946
Wyoming	H. R. Kanable, M.D.	Basin	1945

*Chairman, Board of Governors

Alaska	A. H. Johnson, M.D.	Kodiak	1945
Hawaii	William F. Leslie, M.D.	Honolulu	1945
Philippine Islands	Miguel Canizares, M.D.	Manila	1946
Puerto Rico	Jacob Smith, M.D.	Rio Piedras	1946
U. S. Army	Maj. Gen. S. U. Marietta	Washington, D. C.	1947
U. S. Navy	Capt. Robert E. Duncan	Washington, D. C.	1947
U. S. Public Health Service	Herman E. Hilleboe, M.D.	Washington, D. C.	1946
U. S. Veterans Administration	Col. Roy A. Wolford, M.C.	Washington, D. C.	1946
Canada			
Quebec	J. A. Couillard, M.D.	Mont Joli	1947
Eastern Provinces	A. F. Miller, M.D.	Kentville, N. S.	1947
Western Provinces	George Ferguson, M.D.	Melville, Sask.	1946
Ontario	Harold I. Kinsey, M.D.	Toronto	1946
Argentina	Raul F. Vaccarezza, M.D.	Buenos Aires	1947
Australia	John H. Blackburn, M.D.	Queensland	1945
Chile	Hector Orrego Puelma, M.D.	Santiago	1947
China	Shu-Fan Li, M.D.	Hong Kong	1947
Colombia	Carlos Arboleda Diaz, M.D.	Bogota	1946
Cuba	Octavio Rivero, M.D.	Havana	1945
Ecuador	Juan Tanca Marengo, M.D.	Guayaquil	1946
India	Jaharlal Ghosh, M.D.	Calcutta	1946
Mexico	I. Cosio Villegas, M.D.	Mexico City	1945
Norway	Carl Semb, M.D.	Oslo	1945
Panama	Amadeo V. Mastellari, M.D.	Panama City	1946
Peru	Ovidio Garcia Rosell, M.D.	Lima	1947
South Africa	David Pieter Marais, M.D.	Cape Town	1945
Uruguay	Fernando Domingo Gomez, M.D.	Montevideo	1947
Venezuela	Jose Ignacio Baldo, M.D.	Caracas	1946

COUNCILS AND COMMITTEES

COUNCIL ON MILITARY AFFAIRS AND PUBLIC HEALTH

Charles M. Hendricks, M.D., *Chairman*	El Paso, Texas	1946
Richard Davison, M.D.	Chicago, Illinois	1947
Capt. Robert E. Duncan, U.S.N.	Washington, D. C.	1945
Herman E. Hilleboe, M.D.	Washington, D. C.	1947
Maj. Gen. S. U. Marietta, U.S.A.	Washington, D. C.	1945
Joseph W. Post, M.D.	Philadelphia, Pa.	1946
Samuel E. Thompson, M.D.	Kerrville, Texas	1947
Walter E. Vest, M.D.	Huntington, W. Va.	1946
Col. Roy A. Wolford, M.C.	Washington, D. C.	1945

COUNCIL ON UNDERGRADUATE MEDICAL EDUCATION

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Andrew L. Banyai, M.D.	Wauwatosa, Wis.	1946
Benjamin L. Brock, M.D.	Waverly Hills, Ky.	1947
H. Frank Carman, M.D.	Dallas, Texas	1946
Sydney Jacobs, M.D.	New Orleans, La.	1947
C. Howard Marcy, M.D.	Pittsburgh, Pa.	1945
John H. Skavlem, M.D.	Cincinnati, Ohio	1946
H. I. Spector, M.D.	St. Louis, Missouri	1945
Nelson W. Strohm, M.D.	Buffalo, New York	1945

COUNCIL ON POSTGRADUATE MEDICAL EDUCATION

J. Winthrop Peabody, M.D., *Chairman*	Washington, D. C.	1946
Paul D. Crimm, M.D.	Evansville, Ind.	1945
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Carl R. Howson, M.D.	Los Angeles, Calif.	1946
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Russell S. Anderson, M.D.	Erie, Pennsylvania	1947
I. D. Bobrowitz, M.D.	Otisville, New York	1946
Merle D. Bonner, M.D.	Jamestown, N. C.	1947
Kenneth G. Bulley, M.D.	Aurora, Illinois	1946
J. B. McKnight, M.D.	Sanatorium, Texas	1945
Myron Miller, M.D.	Columbus, Ohio	1945
Joseph R. Morrow, M.D.	Ridgewood, N. J.	1945
Harry Warren, M.D.	San Francisco, Calif.	1947

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James F. Brewer, M.D.	New Bedford, Mass.	1947
D. W. Heusinkveld, M.D.	Cincinnati, Ohio	1945
Robert G. McCorkle, M.D.	San Antonio, Texas	1945
John S. Packard, M.D.	Allenwood, Pa.	1947
S. A. Parowski, M.D.	San Diego, Calif.	1945
John K. Shumate, M.D.	Madison, Wisconsin	1946
Nelson W. Strohm, M.D.	Buffalo, New York	1947

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(Annual Meeting, New York City, June, 1945)

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Note: The following committees have not as yet been completed, and the names of the members of these committees will be published in the next issue of the journal:

Committee to Establish a Board for Diseases of the Chest

Committee on the Management and Treatment of Diseases of the Chest
- Subcommittee on Chemotherapy and Allied Measures
- Subcommittee on General Management and Rehabilitation of Diseases of the Chest
- Subcommittee on Non-Surgical Collapse Therapy
- Subcommittee on Surgical Treatment of Diseases of the Chest

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Juan Carlos Rey, M.D., Buenos Aires, *Secretary-Treasurer*

OBITUARIES

CHARLES HARTWELL COCKE

1881-1944

Dr. Charles Hartwell Cocke, formerly vice-president of our parent organization, the Federation of American Sanatoria, died suddenly at Asheville, North Carolina, on August 3, of coronary disease. He was born December 1, 1881, at Columbus, Mississippi, the son of Charles Hartwell Cocke, president of Mississippi College for Women, and Rowena Lockhart Hudson Cocke. He was the first graduate of the Episcopal High School of Virginia at Alexandria and received his A.B. in 1902 from the University of Virginia. His medical degree was conferred by Cornell in 1905 and he served an interneship at the Presbyterian Hospital in New York, 1906-1907. He then took postgraduate work at the University of Vienna and in London and Paris. Dr. Cocke practiced in Birmingham, Alabama, until he became ill with tuberculosis, when he went to Saranac Lake. Upon his recovery in 1911, he located in Asheville and in 1914 married Miss Amy Grace Plank of Carlisle, Pennsylvania.

He was secretary of the Buncombe County Medical Society in 1917 and 1918, president in 1923 and became an honorary member in 1943. Upon the death of Dr. Charles L. Minor, Dr. Cocke was made governor for North Carolina of the American College of Physicians (1929-1942), chairman of the Board of Governors from 1936-42 and was elected first vice-president in 1942, with reelection in 1944. He was vice-president of the American Clinical and Climatological Society in 1931 and vice-president of the Southern Medical Association in 1932, serving as secretary of its Section on Medicine in 1937. He was certified as a diplomate by the American Board of Internal Medicine in 1936. He was also a member of the Southern Interurban Clinical Club; a member of the American Association of the History of Medicine; American Trudeau Society and National Tuberculosis Association. He was medical director of Zephyr Hill Sanatorium; consultant to Biltmore Hospital, Patton Memorial Hospital, Hendersonville, N. C., Learline Reeves Sanatorium, Greenville, Tenn., and attending physician to Mission and Saint Joseph's Hospitals, Asheville. He was chairman of the local draft board.

Dr. Cocke was the author of numerous papers on tuberculosis and internal medicine, contributing to International Clinics, American Journal of Medical Sciences, Annals of Internal Medicine; American Review of Tuberculosis; Southern Medical Journal; New York Medical Journal; Virginia Medical Journal; New Orleans Journal of Medicine and Surgery; New England Medical Journal; West Virginia Medical Journal and Southern Medicine and Surgery.

"Hartwell" or "Charlie" Cocke endeared himself to a host of friends, patients and colleagues by his accessibility, ready sympathy and genial humor. He combined the courtesy and charm of the traditional Southern gentleman with the alert poise and quick insight of a native New Yorker. He could be depended upon to serve in any worthy cause. As a consultant, his observations were comprehensive and his judgment sound, while his attitude was always fair but frank to all concerned. His writings and addresses had the clarity and force of an experienced teacher. As a presiding officer, he proceeded with efficiency and dispatch. Literature,

travel and golf were his chief means of relaxation in a life which required careful organization to be lived as he lived it.

Karl Schaffle, M.D., F.C.C.P.,
Regent, District No. 4

SAMUEL IGLAUER

1871-1944

After a brief illness, Dr. Samuel Iglauer died at Cincinnati, Ohio, on June 23, 1944. Dr. Iglauer was born in Cincinnati on December 28, 1871. He received his B. S. from the University of Cincinnati in 1895 and his M.D. from the Medical College of Ohio in 1898. Following his graduation from medical school, he spent several years in intensive study in European clinics. He then returned to Cincinnati and practiced otolaryngology until the time of his death.

Dr. Iglauer was a member of the faculty of the University of Cincinnati, as Professor of Laryngology from 1916 until 1930, and as Professor of Otolaryngology from 1930 until his death. He was director of the Otolaryngological Service at the Jewish Hospital, General Hospital and Children's Hospital.

Dr. Iglauer was author of over one hundred papers and did some pioneer work in America in the use of the bronchoscope. He, with Dr. Sidney Lang, was among the first to describe the x-ray appearance of the mastoid bone. He published some of the first articles in this country on the use of lipiodol for studying the diseases of the lungs. He was an authority on the anatomy of the spaces of the neck and gave lectures and published numerous papers on this subject. A recent paper, in conjunction with Dr. William Molt of Indianapolis, described the injury to the larynx resulting from an indwelling duodenal tube.

Dr. Iglauer was a member of the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, the American Broncho-Esophagological Association and a member of the American College of Chest Physicians.

John H. Skavlem, M.D., F.C.C.P.,
Governor for Ohio

FERDINAND CHENIK

1891-1944

After an illness of several months, Dr. Ferdinand Chenik of Detroit, Michigan, died on August 15, 1944, at his ranch near Westlaco, Texas. Dr. Chenik was a native of Austria, and took pre-medical work at the University of Leipzig, Germany. He came to Detroit in 1914, and was graduated from the University of Michigan and Detroit College of Medicine. He owned and was the superintendent of the Chenik Hospital in Detroit.

Dr. Chenik was a Fellow of the American College of Chest Physicians, a member of the American Medical Association, Michigan Medical Association, and the Wayne County Medical Society. He was the author of two books and many articles on tuberculosis.

Dr. Chenik is survived by his wife, Josephine, and three children, Viola, Loretta, and Richard.

THE PHYSICIAN'S IMPORTANCE IN WAR AND PEACE

To memorialize the medical profession's "skill and courage and devotion beyond the call of duty" is the purpose of the new prize-contest recently announced by the American Physicians Art Association.

The contest is open to all physicians, both civilian and military, who are members of the A.P.A.A. The prizes are sufficiently important to attract some very fine art in all of the principal media, including oil, water color, sculpture, and photography.

For full details, write to the association's secretary, Dr. F. H. Redewill, Flood Building, San Francisco, California. Also pass this information on to your physician-artist friends, both civilian and military.

DETROIT REPORTS NEARLY 50 PER CENT DEATHS DUE TO TUBERCULOSIS

Of 44 deaths reported by Dr. Bruce H. Douglas, Commissioner of Health for the City of Detroit, for the week ending August 19, 1944; 32 of these deaths were attributed to tuberculosis (21), lobar pneumonia (5) and broncho pneumonia (6). The other 12 deaths were reported for whooping cough (3), syphilis (4) and poliomyelitis (5). No deaths were reported for 8 other communicable and infectious diseases. There were 79 cases of tuberculosis reported for this week against a norm of 52. Thus, for the week ending August 19, 1944, 73 per cent of the deaths reported for infectious and communicable diseases in the City of Detroit were caused by respiratory disease, with tuberculosis accounting for nearly 50 per cent of the total deaths reported.

NATIONAL DRUG APPOINTS MARTIN RESEARCH HEAD

Philadelphia, Pa.—Mr. A. B. Collins, President of The National Drug Company, announced today that Dr. Gustav J. Martin had been appointed to the position of Research Director of the company. Dr. Martin comes to The National Drug Company from the Warner Institute in New York City where he was Assistant Director in charge of the Division of Chemistry. He is a member of numerous scientific societies and has published more than 50 papers in various scientific journals. The appointment of Dr. Martin is another step toward the fulfillment of the broad research program which National Drug is now undertaking.
